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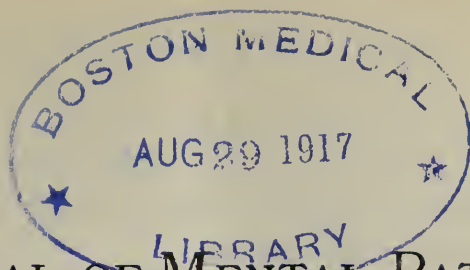
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THE JOURNAL OF MENTAL PATHOLOGY.

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NOS. I AND 2.

ON SOME DIAGNOSTIC DIFFICULTIES IN A CASE OF LESION OF THE SPINAL CORD.

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The difficulty in making a differential diagnosis between an organic and hysterical paraplegia is well familiar to the profession. Indeed, Charcot justly designated hysteria as "the great simulator" of diseases. Another difficulty met with in the diagnosis of transverse myelitis is that of determining the seat and nature of the lesion. The latter may be intra or extra-medullary.

The case I am about to consider presented such difficulties, having drawn some of the leading neurologists into erroneous conclusions. For this reason, the very simplicity of the case presents an unusual practical interest.

The history of the case is as follows: The paternal great-grandfather had senile delirium with delusions of grandeur. He died suddenly—probably of cerebral hemorrhage. The paternal grandfather was a drunkard. He killed himself on account of the death of his brother. A maternal uncle was a drunkard and died of cardiac disease. The father is 50 years of age and is healthy. The mother has been suffering from convulsive spells since she was 18 years of age. These convulsions seem to be of hysterical nature. Aside from this, the mother seems to be healthy. The parents are consanguinously related. The paternal grandfather and maternal grandfather were brothers.

The patient is the ninth born in the family. One of his brothers was operated upon, in 1900, for a tubercular abscess in the sternal region. One sister was operated upon for adenoma (?) of the left breast. She is also suffering from tendo-synovitis of the left

hand that is of tubercular origin. After an attack of a febrile disease his sister, 9 years of age, suffered from hysterical hemiplegia and aphasia during a period of fourteen days. She also suffered from hysterical deafness.

The patient was born under normal conditions after a normal pregnancy. His infancy and adolescence were normal.

The patient, Vincenzo B., was born February 21, 1886. In February, 1902, he went to the theatre C., where a hypnotist exhibited his art of hypnotizing people. My patient took interest in the matter and submitted himself to the operation that proved quite successful in his case; he faithfully enacted the suggestions made to him during the hypnotic sleep. Some time after these incidents, the patient experienced a change in his condition, complaining of weakness in the lower limbs. This weakness gradually increased and finally he became unable to walk. The patient himself conceived the idea that his invalidity was caused by the suggestion made to him by the hypnotist. He had measles and small-pox and his face is pitted. When twelve years of age he had nocturnal incontinence. Puberty developed normally. The patient has not had any sexual relations, and venereal infection may be excluded in his case. Careful study of the case also warrants the exclusion of syphilitic infection by the mouth, etc. In 1900, the patient had an attack of gastric infection, from which he recovered in a fortnight. He never presented any disturbances either of the motor or of the sensory nervous system, save the nocturnal incontinence of urine. He has always been of quiet and gentle disposition and made good progress in his studies at school.

February 7, 1902, he submitted himself for the first time to hypnotic suggestion as related above. According to data collected, the patient was not put into a condition of hypnotic sleep, but was simply brought into a state in which he readily lent himself to suggestion. Thus, it was suggested to him that a tallow candle was bread, and he ate it with gusto. He also enacted the suggested rôle of a drummer, a barber, etc.

During one of these séances, the hypnotizer seated the patient in a chair and, looking him fixedly in his eyes, said: "I dare you to remain seated! You shall have to follow me!"

After the experiment, the patient felt "attracted," as he expresses it. This experiment was made in the above mentioned theatre, before the audience. The patient felt exhausted and went home in a depressed condition. The following morning he remained in bed later than usual, looking pale and depressed and feeling tired. He said that he felt as if he had been flogged and had spent a sleepless night. He finally dressed himself, but felt

depressed and tired during the day. The second day after the experiment he said to a friend: "I feel exhausted and have a pain in the left side." Five days after the experiment the pain became more generalized and intense, attacking particularly the "back," as the patient expressed it. In reality, however, the pain was felt in the left chest, lumbar, dorsal and sacral regions of the back. There were no pains in the arms, legs or waist. These pains spread forward, and there was even a question as to the patient suffering from left pleurisy and pericarditis. There was no elevation of temperature, however.

Although the patient stooped when walking, on account of the pains, the latter were neither continuous nor intense. He preferred to remain out of bed because when he was in a horizontal position he suffered more from the pains, could not breathe and spent sleepless hours. This condition lasted for some two months after the experiment. Then, in May, he began to suffer from tingling sensations in both lower limbs, whether in the upright position, seated or in bed. Other disturbances of sensibility also manifested themselves. Thus, he felt as if he were walking on cotton, and unless he looked on his feet, he could not tell whether he had his shoes on or off. Besides, the limbs became weak and he had to walk with a stick or be led by an attendant. If he tried to walk fast, he felt as if he were being held back, stumbled and even fell.

The tendon reflexes of the lower limbs were exaggerated at this period and there was spinal trepidation. At the same time there was probably increased muscular tonicity. The patient also suffered from frequent erections that were caused by the slightest touch of the clothes or hand. The erections were not accompanied by any sexual desire. At rare intervals he had nocturnal pollutions, but never diurnal. Later on he began to suffer from constipation and bladder disturbances. The constipation became troublesome, the patient passing some ten days at a time without having any bowel movement. The bladder disturbances also became troublesome. There was difficulty in urination that was accompanied by vesicular tenesmus, that sometimes lasted an hour before the act of urination could be accomplished. Frequently there was erection during the spells of tenesmus.

Meanwhile paraparesis of the lower limbs set in and within some ten days the patient had to be confined to bed on that account. This was accompanied by disturbances of sensibility. The patient felt as if the upper and inner part of the thighs were unusually enlarged. And while the motor disturbances were at first equally distributed on both sides of the body, they now became more

marked on the left side. Thus, motility in the left lower limb was now completely lost, while the right one could still execute limited flexion and extension movements. Later on this motility was also completely lost and the patient then suffered from complete paraplegia. The bladder disturbances also became more marked. The patient remained in bed during the months of May and June. The patellar reflexes were still exaggerated and there was hyperesthesia of the lower limbs. The abdominal muscles now became the seat of marked weakness. The patient could not assume a sitting posture without the help of an assistant, and when once seated, could not keep the position without being held by the shoulders.

During the first part of June the patient had a convulsive attack. When the physician was about to apply the Paquelin thermocautery, the patient became excited and had a typical hysterical attack, accompanied by various motor and mimic disturbances of the body and limbs, including the rainbow attitude. There was no loss of consciousness, biting of the tongue, foam at the mouth or loss of urine. The attack lasted two hours. He had a similar attack every day for the following three days. June 27, cauterization was applied in the dorsal and lumbar regions of the spine. June 29, there was a more severe attack than those described and it was accompanied by loss of consciousness. Immediately preceding the attack, the patient, who was unable during the preceding month either to sit up or hold himself in the sitting position, suddenly sat up, called his mother, complained of a sensation of strangulation rising in his throat and fell in the attack that lasted about an hour. There was involuntary urination during the attack and complete amnesia of the occurrence after the attack.

After this attack, the patient manifested complete loss of control of the urine and feces. The incontinence was preceded by retention. Some time after, the dripping of the urine stopped, but the paralytic manifestations of the bladder with retention remained. This trouble was aggravated by a muco-purulent cystitis caused by the frequent use of the catheter.

In July, the hyperesthesia of the lower limbs disappeared, making room for anesthesia. There was decreased reaction to faradic and galvanic currents in the lower limbs. During the months that followed the patient manifested various disturbances. Thus, he had attacks of deafness and aphasia of five minutes' duration, at various intervals. At times these attacks were more or less prolonged. At times he saw objects double.

Various treatments had been tried but without results. Medication, injections of bichloride of mercury, electricity and sugges-

tion—all were in vain. Although the patient was a most submissive subject the night when the hypnotist first experimented on him in the theatre, he remained rebellious to hypnotism and all possible treatment at present. He was finally taken to the country and isolated from his relatives. Here he underwent the treatment of massage and metallo-therapy.

According to data furnished me, all the forms of sensibility of the lower limbs reappeared, but the pelvic area remained anesthetic. The physician in attendance found it impossible, however, to make the patient walk. The only point gained in the matter of improving motility was as follows: when held by assistants in an upright position, he could drag backward one lower limb that had purposely been put forward. The question of this apparently improved motility will be discussed later on.

At the request of the patient himself, the physician tried to hypnotize him, but without any results. When the patient returned home, May, 1903, sensibility was completely absent in the lower limbs. June 9, he suddenly lost consciousness and remained senseless for about an hour. The limbs were cold, the face pale and there were some twitchings about the mouth. He then came to himself and soon fell asleep. He woke up without showing after-effects.

Some time later, he had an abscess in the left axilla with some febrile manifestations. November 2, pleurisy set in on the right side and later on there was effusion. Two litres of limpid liquid was drawn, and the patient recovered from the pleurisy.

Summary of the principal features of the case.—February, 1902, experimental hypnotic suggestion. March and April, pains along the spine. May, paresthesia of the lower limbs. Finally complete paralysis of these limbs. Bladder and rectal disturbances ranging between paresis and complete paralysis of the organs. Later on, convulsive manifestations, spells of deafness and aphasia lasting from five minutes to one-half hour. Right pleurisy with effusion. Therapeutic attempts made in vain.

OBJECTIVE EXAMINATION.—I examined the patient for the first time August 21, 1903. He was rather well formed, somewhat undersized and the face and mucous membranes were pale. The cranium was oval shaped and slightly flattened in the left frontal and right occipital regions (plagiocephaly). So that the left oblique diameter was 180 mm., and the right oblique diameter 188 mm. The teeth were normally implanted, save the lower incisors that were imbricated and inclined towards the right. The hair was light brown and fine, implanted somewhat low on the

forehead. The eye-brows were well formed and united at the glabella, the eyes were dark-brown and the sclerotic bluish. The pupils were round and central, the nostrils somewhat large. The breasts were abnormally developed for a male subject and the case was typical of gynæcomasty. There was nothing of note in the configuration, except for an S-shaped curvature of the spine, the concavity of the upper branch being directed to the left and that of the lower to the right. The physiological anterior curve was also exaggerated. Thus, as is seen from the facts stated above, there was nothing abnormal in the configuration of the spine, as a perfectly symmetrical spine is hardly ever found. In the left axilla was the tumor, about the size of a mandarin. Most probably the growth was a diseased lymphatic gland or a periadenitis.

The right chest was filled with fluid up to the scapula. An exploratory puncture showed that the fluid was straw-colored and of sero-fibrinous nature. There seemed to be about two litres of fluid in the chest, but the heart seemed to remain in its normal position and the cardiac beats were normal. The liver was somewhat pushed downward, offering an area of about two fingers' breadth below the costal line. There was no tenderness on palpation. There was retention of urine and constipation. The bladder was emptied twice a day. The patient was slightly feverish. There was slight cutaneous edema of the lower limbs that was particularly noticeable about the ankles and dorsum of the feet. The skin was cold and desquamated easily.

The urine analysis gave the following results: Specific gravity, 1022. Turbid, color deep red, reaction acid, deposits amounted to 6.50 per cent. No albumen, sugar or pus. Abundance of alkaline phosphates and of chlorides. Minimum amount of carbonates. Excess of urates.

DISTURBANCES OF SENSIBILITY.—There was hyperesthesia of the left parietal region. There was a special sensation of cold in the feet when they were exposed to the air for any length of time. The patient felt as if the feet had been plunged into cold water. Objectively, however, the feet were normally warm. There was cutaneous pruritis of the left abdominal and lumbar regions whenever he filthied his bed. Through this sensation and the odor of the feces only did he know that he had filthied his bed. The rectal anesthesia and that of the nates seemed to be complete, so that he did not feel under him either the fecal matter or the urine that he passed involuntarily.

During the spells of deafness and aphasia there was impaired sensibility and a creeping sensation of the lips. When the spell was over, these sensations disappeared on the left side of the lips

but persisted on the right. These sensations also involved the neck, right shoulder and right upper limb down to the tips of the fingers. The sensations lasted as long as did the spell, for a period of from five to fifteen minutes. During these spells the patient also had a sensation of a ball rising in his throat. Cranial percussion was negative, except in the temporal zone, where there was hyperesthesia, as related above. Pressure along the spine caused a disagreeable sensation, especially in the interscapular region. Pressure of the nerve trunks and the muscles gave negative results. Pressure of the xyphoid end of the sternum, on the contrary, was painful.

The upper limbs and upper part of the body beginning with the waist line did not present any permanent disturbances of sensibility. Sight, hearing, smell and taste were normal. The taste of salt only was impaired. There were grave disturbances of sensibility, on the contrary, in the lower part of the body. There was a generalized cutaneous anesthesia of the entire lower part of the body, including the inner part of the pelvis and the perineum. The upper line of demarkation of this vast area of anesthesia was about as follows: Anteriorly—up to the xyphoid process of the sternum, posteriorly—to the last dorsal vertebra and a bit lower than this level over the hips. The disturbances in this vast area were those of touch, heat, cold and pain. The anterior line of demarkation was almost horizontal and stationary, while the posterior line often varied even during one and the same examination, within the limits of from 5 to 8 centimetres higher or lower than indicated above. Above the anesthetic zone there was a limited zone of hypoaesthesia. As has been remarked, there was also vesicular, rectal and testicular anesthesia.

Tactile and heat stimuli of the lower part of the lower limbs, both of the right and left side, were always referred to a limited area, about the size of the palm of the hand, in the middle third of the left thigh. Stimuli of pain and cold were not thus referred to the area in question. Electro-cutaneous and electro-muscular sensation was abolished. When the muscular masses of the limbs were handled, the patient had a vague sensation of being touched, but could not always correctly locate this sensation. The sensation of position of the lower limbs was impaired, the patient having no idea of their position when his eyes were closed.

The anesthetic zones were also ischemic, pricking with a pin being accomplished without any bleeding.

MOTOR APPARATUS.—The ocular muscles were normal. The muscles of the face, neck, upper limbs, thorax and back were

in normal condition. There was normal muscular tonicity and the dynamometer registered 39 for the right and 28 for the left hand. The movements of the upper part of the body and upper limbs, as outlined above, were normal. The abdominal muscles, on the contrary, presented a certain degree of paresis. The marked meteorism pointed towards this fact, as also did the difficulty the patient experienced in assuming a sitting posture.

In the lower limbs the muscles were flaccid, their tonicity was impaired and activity abolished. The limbs were like dead masses and did not react to any stimuli or movements. The same was true of the genital organs, rectum and bladder. Electric reaction was absolutely abolished, even to the galvanic current.

REFLEXES.—The pupils reacted normally to light and accommodation. The conjunctival, nasal and oral reflexes were normal. The masseter, auricular, axillary, mammary and scapular reflexes were normal. The tendon reflexes of the arm and forearm were rather limited. The same was true of the biceps, triceps, ulnar and radial reflexes. The epigastric, abdominal, cremasteric, anal, perineal, gluteal and plantar reflexes were abolished. All the deep reflexes of the lower limbs were abolished, including the patellar, Achilles, etc.

VASO-MOTOR DISTURBANCES.—There was edema of the lower limbs, especially of the dorsum of the feet. This edema became more marked when the patient was in a sitting posture. Pressure in the edematous area left pitting that persisted for four days, even if the patient remained in bed. There was anemia of the anesthetic areas. Dermographism was quite marked on the chest.

The muscular atrophy of the lower limbs can be judged from the figures in the table below.

MEASUREMENTS OF THE LOWER LIMBS IN 1903.

	Ten cm. below the patella. cm.	Fifteen cm. below the patella. cm.
September 28, right	42.7	31
September, 28, left	42.5	31.5
October 28, right	39	31
October 28, left	39	31.5
November 6, right	37	30
November 6, left	36.5	30
November 12, right	38	28.5
November 12, left	37	28.5

The following were the characteristics of a spell that was followed by so-called aphasia.—The patient suddenly became pale, looked fixedly into distance and proffered some incoherent syllables or words. He held his head inclined to the right and said: "This is enough! Enough, enough!" He remained in this condition of hebetude for a few minutes and then said: "I feel better now." When asked some questions, he answered: "Let me alone. I cannot understand you." Hence, it seems that there was no deafness and aphasia in the true sense of the word. The patient's own words are significant in this respect. He said: "I may be able to understand you, if you speak slowly, syllable by syllable. Otherwise I forget your words as soon as you have spoken them and, therefore, cannot answer you."

It seems that during these spells the auditori-verbal centre was in a paretic condition that was probably caused by vaso-motor spasm. The accompanying creepy sensation and anesthesia, as explained above, speak in favor of vaso-motor spasm in the following convolutions: the first temporo-sphenoidal (verbal-auditory memory) and the foot and middle of the Rolandic convolution. It seems reasonable to suppose that this abnormal stimulus spread through the entire ascending frontal and parietal convolutions. This reasoning would be more warranted and even supported if the right lower limb had been in a condition to feel and if there had been in this limb a creepy sensation during the attack.

On the psychic side there was nothing of note. The patient generally had frightful dreams.

Last April, his condition became quite worse. The muscular atrophy of the lower limbs was very much increased, the temperature varied between 38.5 and 39 degrees C. Pulmonary tuberculosis rapidly ran its course and the patient died in March.

GENERAL CONSIDERATIONS.—The history of the patient leaves no doubt that he was subject to hysteria. The question of interest, however, is whether the paralytic symptoms were of functional or organic nature. From a clinical point of view this question is of grave importance. It seems reasonable to consider whether this case presented a combination of hysterical stigmata with organic paraplegia. As has been shown, the main features of the case were as follows:

Tubercular heredity. The disease began first with pains, then followed paresthesia and spastic paraparesis, and finally there was flaccid paraplegia. There was tenderness of the spine on pressure. Incontinence of urine and feces. Loss of reaction to the faradic and galvanic currents. Comparatively rapid development of

muscular atrophy in the affected parts. Erection without sexual appetite. Subsequently there was complete abolition of sexual desire. The motor disturbances preceded the sensory disturbances.

All these facts speak in favor of an organic rather than of a hysterical paraplegia. In hysterical paraplegia there is hardly ever any bladder and rectal paralysis. The muco-purulent cystitis speaks in favor of an organic affection. The involuntary loss of urine and feces in this case is characteristic of an organic paraplegia. Hysterical subjects, on the contrary, are conscious of the evacuation of the bladder and bowels. The muscular atrophy in this case developed early and rapidly. In hysteria, on the contrary, the onset of such atrophy is late in the disease and slow of development. In this case there was absolute abolition of electric excitability, both to the faradic and galvanic currents. In hysteria, on the contrary, such decrease, if it exists at all, is only slight in degree. Abolition of the tendon and skin reflexes and anesthesia may exist in both instances. Considering the other symptoms of the case, however, it is reasonable to admit that these disturbances were rather of organic than of hysterical nature. The facts speaking in favor of the organic nature of the anesthesia and paraplegia are: the pains, hyperesthesia, torpor and tingling in the lower extremities that preceded the paralysis. Such grouping of disturbances is difficult to find in hysteria. Besides, hysterical anesthesia, when involving a limb, generally presents a line of demarkation on a level with the root of the limb. In this case, on the contrary, the line of demarkation did not speak in favor of any hysterical disturbance.

I mentioned the fact that the patient, while being paralyzed, succeeded in dragging backward his leg while he was supported by an attendant. In reality, however, the patient must have lifted up the whole body, and the limb dragged itself by sheer force of its own weight. It was noted above that during the convulsive spells the patient made some slight movements with the lower limbs. It should be borne in mind, however, that this took place in the beginning of the disease, when the paralysis was not yet complete. The great variability of objective sensibility may be explained by vaso-motor disturbances, due to stasis, edema and hyperemia, caused by the major lesion.

Taking all the facts into consideration it seems to be reasonable to suppose that the patient suffered from paraplegia of an organic nature.

The absence of involvement of the cranial nerves and of cere-

bral disturbances excludes all possible cerebral involvement. Paraplegia of neuritic origin may also be excluded on account of the absence of tenderness to pressure in the peripheral nerves of the lower limbs. And the presence of the bladder and rectal symptoms, generally speaking, also exclude peripheral neuritis. It seems only reasonable to suppose that there was a localized spinal lesion.

It seems reasonable to locate the lesion in the lumbar region of the spinal cord. The facts speaking in favor of this localization are: the flaccid paraplegia with abolition of the cutaneous and tendon reflexes, cutaneous anesthesia reaching up to the level of the xyphoid process in front, the bladder and rectal paralysis, loss of sexual desire and, finally, the muscular atrophy of the lower limbs. If the zone of anesthesia indicates the level of the lesion, it may be supposed that the lesion involved not only the lumbar but also the dorsal region in its lower part. The clinical facts seem to correspond perfectly to a chronic transverse lumbar myelitis. It is difficult to say, however, whether the lesion involved the spinal cord itself or that it was extramedullary of syphilitic, tubercular, pachymeningeal or other nature.

It is reasonable to suppose that in this case the affection was of tubercular nature. The patient's family history and his own tubercular affection speak in favor of this supposition.

It is interesting to consider whether the tubercular lesion, that we suppose existed in this case, was intra- or extra-medullary. It seems that tubercular spondylitis may reasonably be excluded. Indeed, vertebral caries generally take place in the dorsal, sometimes in the cervical region, but rarely in the lumbar region. In my case repeated examination failed to reveal any kyphosis in the lumbar region. It is true that caries may exist without causing evident kyphosis. The other characteristic signs of caries, however, were also absent. Thus, there was no rigidity of movement in any part of the spine or head, nor was there any exaggerated tenderness or pain on pressure along the vertebræ. As is well known, these symptoms hardly ever fail, even when the deformity is absent. The intense and persistent pains characteristic of Pott's disease were absent in my case. The only pains noted were the slight uneasiness in the left side and in the back in the beginning of the disease. In my case paraplegia and disturbance of sensibility was complete, while in tubercular spondylitis the motor symptoms alone are predominant and the sensory sphere is only slightly disturbed. In my case the symptoms were equally distributed on both sides, while in tubercular spondylitis a predominance of the symptoms on one side is the rule. It seems

reasonable to exclude Pott's disease in my case, because the disease could hardly have run its full course, until death took place, without having manifested any of its proper characteristics.

Spinal pachymeningitis may also be excluded, as its characteristics were absent. In my case there was no symptomatology of irritation of the posterior roots. There was no paresthesia or hyperesthesia accompanied by continuous irradiating pains of neuralgic coloring. Nor were there any localized dorsal and sacral pains that increased on movement of these regions or on pressure of the vertebræ in the same. On the contrary, in my case the pains were so slight that the patient hardly noticed them. In pachymeningitis the sensory disturbances may be accompanied by atrophic paralyses of given groups of muscles. When the spinal cord is also involved there may be added paretico-spastic phenomena in one or both lower limbs. The clinical picture in my case, however, was quite different. Involvement of the entire breadth of the spinal cord is also a rare occurrence. Generally only part of the cord is involved, giving the Brown-Séquard syndrome, that was absent in my case.

Symptoms of an extramedullary tumor were not present in my case. Indeed, a tubercular or any extra-medullary tumor is characterized by two sets of symptoms: 1, segmentary; 2, those of interruption of spinal conduction. If the tumor encroaches on the posterior roots, "symptoms relating to the spinal roots" are manifested from the very beginning. These symptoms are: irradiating pains in the zones of distribution of the nerves, the roots of which are affected. The pains may be either persistent and burning or intermittent and neuralgic in character. The manifestation may also be expressed by paresthesia or anesthesia of radicular type. If, on the contrary, the tumor involves the anterior spinal roots, there are symptoms of motor irritation from the very beginning and they are followed by muscular paralyses and atrophy in the respective areas. In some cases of such tumors the manifestations commence with paralysis of one muscle. Professor Mingazzini recently published such a case, in which a tumor of the body of a vertebra caused, as a first symptom, paralysis of the serratus muscle. The atrophy that followed confirmed the diagnosis. The segmentary symptoms naturally extend with the spread of the tumor—upwards or downwards. Interrupted spinal conduction varying with the seat of the lesion naturally follows when the spinal cord is involved. It may be accepted in general that the presence of paraplegic symptoms points towards spinal involvement. Thus, then, there were no signs of involvement of the medullary roots, such as irradiating and persistent

pains accompanied by a burning sensation, neuralgic spells, paresthesia and anestheisa of radicular nature, etc.

By exclusion, it remains to consider the possibility of an intramedullary tubercular tumor. According to Oliver, scrofula may be a cause of myelitis. Rokitsky says that it is rather rare to find tubercular masses in the spinal cord. The tubercular infiltrations that had come to his notice were located in the lumbar and cervical regions. Lebert also thinks that tubercular lesions involve most frequently the lumbar region. According to Hesse, tubercular infiltration of the spinal cord may be caused by caries of the vertebræ, basillar meningitis, etc. Tiouville published an important work on this subject in 1869, showing that not only the meninges, but also the connective tissue of the spinal cord infected with tuberculosis may cause tubercular growths of the spinal cord. Among these conditions he distinguishes tubercular meningitis, tubercular meningo-myelitis and tubercular myelitis. Raymond distinguishes two groups of tubercular myelitis. Chronic myelitis, characterized by one or more tubercular foci with secondary degeneration. Acute myelitis, that may be infiltrated or nodular in form. These two acute forms almost always co-exist and are accompanied by meningeal lesions and generalized cortical leptomeningitis. Haskovec thinks that the nodular form may be acute, sub-acute or chronic, and supports this view by some clinical and anatomical studies.

In my case there is every probability that the lesion was of the disseminated type, and was located in the lumbar and lower part of the dorsal region. As for the finer traits of the diagnosis, it is impossible to discuss them in my case. We may only say with Leyden that tuberculosis of the spinal cord may present symptoms simulating either those of a tubercular tumor or of acute myelitis of the spinal cord. It is difficult to make a positive diagnosis in these cases. Philippe and Cestan, for instance, found in a case of Pott's disease of the lower dorsal region various foci of disseminated sub-acute parenchymatous myelitis along the entire height of the dorso-lumbar cord. There was neither pachymeningitis, vascular or interstitial involvement.

I can only repeat with Leyden that it is most difficult to diagnose tubercular myelitis of the spinal cord.

JUNE 10, 1904, ROME, ITALY.

TWO CASES OF FAMILIAL HEREDO-SPINAL
ATROPHY (FRIEDREICH'S TYPE) WITH
ONE AUTOPSY, AND ONE CASE OF
SO-CALLED ABORTIVE FORM OF
FRIEDREICH'S DISEASE.

ANATOMOPATHOLOGICAL AND CLINICAL STUDY.

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and

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I.—CLINICAL CASES.

The study of the neuropathology of Friedreich's disease is certainly of the utmost importance, and within the last thirty years much valuable material has been contributed towards its elucidation. While the analytical and synthetical conceptions of the disease differ with the individual authors, a general anatomopathology of the affection is accepted by all. It now remains to come to a uniform understanding as to the relation of the syndrome to its anatomopathology. Broadly speaking, the various studies of the disease have only led up to the conception that the anatomopathology consists of atrophy and agenesis of the spinal cord in its relation to the rest of the central and peripheral nervous systems.

We have had occasion to study two cases of familial heredo-spinal atrophy or Friedreich's disease and one abortive form of Friedreich's disease at various periods of its development. The comparative rarity of these cases and the autopsy of one of them warrants, we think, our presenting a somewhat detailed account of our study in question.

Case I—Maria X., 23 years of age. The paternal and maternal grandfathers were second cousins and married two sisters, who were the patient's paternal and maternal grandmothers. The paternal grandmother died of cardiac paralysis. The patient's parents are cousins. The father is an inveterate drunkard. He is free from syphilis. Two paternal aunts are alcoholists. One paternal aunt died of convulsions during infancy. Two aunts are highly nervous and one aunt is insane. One of two boys of the latter aunt is rachitic. One maternal aunt died in an asylum, two uncles are alcoholists, another uncle suffers from "tic." Three of his children were prematurely born, were hydrocephalic and died during infancy. A physician's diagnosis of these cases was "familial cerebellar affection." One of the two uncles who were alcoholists had two children, who died of eclampsia.

Maria was born when her mother was twenty-one years of age. The patient was the first of seven children in the family. The second child died of some unknown disease. The third pregnancy ended in an abortion. The fourth child was a boy and he died of Friedreich's disease at the age of twenty years (see above). The fourth child is hydrocephalic, but seems to be in good physical condition. The sixth child died of eclampsia, and, finally, the seventh child is healthy. The patient, Maria, was born a few days before term, but the confinement was otherwise normal. Her early childhood was quite normal. At the age of eight years she fell from a considerable height. Although she was dazed by the fall, neither the head nor the spinal column sustained any injury. The first symptoms of her present disease appeared at the age of ten years. At that time, both the patient and her parents noticed that the child's gait was impaired. This disturbance of gait was not preceded by any pains whatsoever. A few months later, the child's handwriting became defective, the tracings of the letters being irregular. The school teacher reprimanded the pupil, thinking that the defect was due to negligence. At the age of twelve years, according to the parents, the child presented deviation of the body when walking. One year later she began to stumble and often fell while walking. She continued to go to school, however, until she was 14 years of age. She menstruated for the first time at that period. The succeeding menstrual flows were either scarce, irregular, or too prolonged. The motor disturbances and bodily feebleness were gradually increasing, and after the age of sixteen years the patient was unable to hold herself on her feet. Synchronously with these symptoms, according to the parents, the patient presented tremors of the hands at various intervals and her speech became exceedingly slow. During the entire course of the disease the patient did not present any pains, properly speaking. She only complained of some vague sensations of uneasiness in the lower limbs in particular. Repeated questions put to the patient and inquiry into the subject warrant the conclusion that she was free from lancinating pains. Various physicians had prescribed for her strychnine, tonics, electricity, but no results were obtained. She was finally examined by us January 20, 1899.

The movements of the ocular clobes were perfect. There was nothing of note in regard to the facial or lingual nerves. The active and passive movements of the upper limbs were good. Muscular force was rather good. There was no tremor of the hands when extended. There was nothing noteworthy in regard to the trophic condition of the muscles

of the upper limbs. The lower limbs were rather underdeveloped for the age of the patient. The muscular tone was normal. The legs were somewhat bent, their curve looking inwards. When extended in bed, the patient's feet hung passively over the edge of the bed and were turned inwards, the disturbance being more marked on the left side. The arch of the feet was normal on both sides. There was hyperextension of the toes on both sides, but it was more marked on the left. Passive movements were enacted without there being any resistance. Muscular tonicity was decreased. The movements of the thighs were somewhat limited on both sides. Flexion of the legs was more limited than was their extension. Movements of the left foot were completely abolished. The movements of the right foot were limited to dorsal flexion. The muscular force of the thigh and leg was somewhat decreased and that of the foot was completely abolished. The reflexes of the upper extremities, as well as the patellar reflexes, were absent. The pupils were equal and of medium size. The iris reacted spontaneously to light and accommodation. Tactile sensibility was decreased in both feet. This sensibility became more evident the more distant the part examined was from the feet. The same was true of the sense of pain. The sense of heat was present, but was slightly decreased in the areas mentioned. The appreciation of cold was normal. There was considerable difficulty in putting the tips of the index fingers in apposition, both with the eyes open or shut. There was a certain amount of tremor of the hands when grasping a glassful of water. There was no tremor, however, when the glass of water was held in the hand. Pressure of the peripheral nervous trunk was painless. The same was true of the pressure along the nerves of the spinal cord. There was no disturbance either of micturition or defecation.

Vision was normal on both sides. Color sight was normal and the visual field was normal.

Speech was distinctly scanning. The patient could not walk without being supported on both sides; the legs diverged one from the other. When made to walk while supported by assistants, the heels were off the ground, while the large toes dragged on the ground.

The handwriting was irregular and the tracing of the letters tremulous.

Since January, 1899, there is nothing in particular to be added in regard to the course of the disease. The parents state that in the early part of the year they first noticed that there was a continuous tremor of the head. The tremor gradually disappeared, while the already limited motility of the lower limbs became more and more impaired. Within the last five years the patient has been confined to her bed or armchair. The parents state that the patient frequently falls on her back while sitting in bed. It is difficult, however, to ascertain the nature of these falls.

STATUS PRÆSENS.—The skin is pliable and pallid, the veins are rather superficial and prominent. The hair is light, thin, lustrous and fine. The insertion of the hair is like that in man. The eye-brows are rather thick and unite at the root of the nose. The axillæ and pubes are quite well covered with hair. The rest of the body is devoid of hair. The head is somewhat small, of ovoidal shape, without there being any plagiocephaly. The type of the face is that between a triangular and ovoidal. There is no plagio-

prosopia. The forehead is sufficiently high and broad in proportion to the rest of the face. The eye-balls are of medium size. The blue of the iris does not tend towards bichromatism. The pupils are normally shaped. The nose is long, the nostrils delicate and the septum regular. The lips are rather thin and the mouth of medium size. The ear-shells are well formed, well distanced from the head, and at an even height and the lobules are adherent. The *processus rami mandibularis* are not marked. The dental arcades are normal, their apposition is normal, the insertion of the teeth is normal. The four wisdom teeth are missing.

The vault of the palate is sufficiently arched, although somewhat narrow. The facial profile is orthognate and the chin is normal. The thorax is rather of a cylindrical form. The breasts are rudimentary, flabby, but normally inserted. The gluteal muscles are rather wasted. The wrists and ankles are slender. The furrows of the palms of the hands seemed normal. The relative length of the fingers is normal. The thenar and hypothenar eminences are rather wasted. The finger nails are small.

PHYSIOLOGICAL NOTES.—The patient is of slender form. Her general physical development seems to be below par in regard to her age. She appears about ten years younger than she really is. The defective development is not accompanied by any rachitic symptoms of the epiphyses. The only noticeable point in this regard is a slight degree of tibial curvature. There do not seem to exist any signs of hereditary syphilis. The muscular and adipose tissues are rather wasted. The skin and mucous membranes are pallid. The cardiac area is normal. Auscultation reveals a soft and characteristic murmur of anemia. The pulse is rhythmical, easily compressible but of normal rapidity, beating between 75 and 80 beats per minute.

The respiratory apparatus seems to be in normal condition.

The tongue is clean and the intestinal function normal, although there is some slight tendency to constipation.

The function of the bladder is normal.

The lymphatic glandular system is normal, save for some enlarged inguinal glands, varying in size between a hemp seed and a small pea. There is no edema about the ankles. The eye-lids are slightly swollen. Both feet present on their external sides of the dorsal surfaces some slight solution of continuity of the skin. The abrasions are irregular in outline, the lips of the abrasions being marked by old cicatricial tissue.

The cicatrices seem to be traces of old abrasions.

There was neither sugar nor albumen in the urine. The latter was simply turbid from an abundance of sediments.

NEUROLOGICAL NOTES.—The aperture of the eye lids seems to be normal on both sides, although the slight edema renders the opening somewhat insufficient. Constriction of the palpebræ is accomplished with some slight difficulty on account of the edema mentioned above. Frowning is also accompanied by some slowness and difficulty. The ocular movements are normal. There is no nystagmus even during extreme lateral movements of the eye balls. Slight and slow nystagmiform movements are noticed, however, when the patient looks into an indefinite distance. During repose the right naso-labial furrow is more effaced than the left one. When the patient grins, the right corner of the mouth is contracted less energetically than is the left one. It cannot be said of the right angle, however, that it is lowered. The movements of the lips are normal and they present no tremors. The other peri-oral muscles are also free from tremors. There is rather a slight depression in the motility of these muscles. It is difficult to know exactly the reason of the hypotonia of the VIIth nerve on the right side, inferiorly. Most probably, the disturbance is due to some slight functional asymmetry. Indeed, there is no accompanying disturbance of motility whatsoever of the tongue, the latter presenting no deviation whether in *situ* or when protruded. Generally speaking, there is no tremor of the head as a whole. There is rather that special appearance that the French authors call facial nystagmus. Of the abnormal movements, besides the slight fibrillary movements of the tongue, there are slight tremors of the muscles innervated by the cranial motor nerves. Thus, there are slight tremors of the nostrils, the peri-oral and peripalpebral muscles. On careful examination it may also be observed that the head and the body are subject to slight rhythmical motor shocks akin to the movements commonly used to enact affirmation or negation. These movements are particularly noticeable when the patient changes her position. When she is in a prone position these movements cease. The upper limbs do not present any particular movements or positions. Although the muscular masses of these limbs are wasted, generally speaking, they do not present any absolute or relative hypotrophy. The trophism of the upper limbs may be judged from the following table of mensurations :

	m.m.		m.m.
Arm, right, upper third.....	143	Left	143
Arm, right, middle third.....	174	Left	168
Arm, right, inferior third.....	216	Left	210
Forearm, right, superior third.....	206	Left	201
Forearm, right, middle third.....	245	Left	240
Forearm, right, inferior third.....	254	Left	251

Passive movements of the upper limbs are accomplished equally well on both sides without any abnormal resistance. Active movements are equally well accomplished by both upper limbs. Although the finer movements, such as apposition and opposition of the index finger with and to the other fingers, are accomplished somewhat slowly, they are still accomplished creditably. The hands are slightly tremulous when in action, such as carrying a glassful of water to the mouth. This tremor is so slight, however, that it can hardly be characterized as an intentional tremor. Reaching out for an object at a distance, the patient does not execute the movement abruptly, but in a normal manner, except for the slowness mentioned above. With her eyes open she could bring the tips of her index fingers into apposition without any difficulty. With the eyes closed she had some difficulty in accomplishing the same act. The defect was so slight, however, that it can hardly be called abnormal. The muscular force was weak on both sides, registering less than five of the dynamometer.

THE LOWER LIMBS.—When the patient was on her back, the calves of the legs were turned inwards, so that both internal margins of the calves touched the sheet and both feet were crossed. The muscular hypotrophy of the lower was more marked than was that of the upper limbs. The degree of the hypotrophy was similar on both sides. The hypotrophy is illustrated in the table that follows.

	m.m.		m.m.
Thigh, right, superior third.....	440	Left	438
Thigh, right, middle third.....	345	Left	343
Thigh, right, inferior third.....	293	Left	293
Leg, right, superior third.....	240	Left	239
Leg, right, middle third.....	231	Left	230
Leg, right, inferior third.....	165	Left	165

The great toes were hyperextended, the extension being more marked on the right. The plantar arch was abnormally accentuated, but not to a maximum degree. In a word, the feet presented a characteristic *equino-varus*. The muscular tissues were flaccid. Passive movements were partly possible on both sides. The resisting power, however, was more marked on the right than on the left. On both sides also the resisting force is less in the thighs, more marked at the knees and maximum at the feet—where dorsal flexion is impossible, while rotation can partially be executed. The passive movements of the thigh over the pelvis, properly speaking, were normal on the left, while on the right side there was a slight increase of resistance to flexion.

Active motility of the lower limbs was almost nil, being reduced to minimal rotary movements of the feet. Even when the position of the legs was corrected mechanically, the stereotyped vicious position was resumed a few minutes later. This defect is even more marked on the right side. Thus, when the patient tried to move the limb while she was in bed, she could not lift it up, the act ending in contraction of the muscles of the trunk and the pelvis. When one of the limbs was lifted up for her, she was able to maintain it for a few seconds in the position in which it was put. The limb then limply fell upon the bed. When one of the limbs was lifted up so that the operator could slip his hand under her knee, the patient could make some slight movements of extension, while those of flexion were nil. This limited extension was accomplished more easily on the left side.

The patient was absolutely unable to use her feet. When helped by two assistants to assume an upright position and asked to take some steps, she let herself be led by the assistants, her feet dragging on the ground as explained above, the limbs remaining in the stereotyped position. It was impossible, under these conditions, to ascertain whether Romberg's sign existed.

REFLEXES.—The pupils were equal, of medium size and of regular contour. They reacted well to light, accommodation and dolorific stimulation. The corneo-conjunctival and pharyngeal reflexes were diminished on both sides. The abdominal reflexes were sufficiently marked on both sides. The axillary, gluteal and popliteal reflexes were absent. The following reflexes were also wanting on both sides: the deep reflexes of the upper limbs, the acromial, bicipital, tricipital, ulnar, radial, radio-carpal and those of the hypothenar eminence. The patellar and Achilles reflexes were absent. Babinski's sign was obtained by tickling the sole of the foot. This sign was more distinct on the right side. Dermographism was wanting.

SENSIBILITY.—Repeated examinations warrant the statement that the tactile, thermic and dolorific sensibility was intact. This sensibility was equally preserved on both sides. The various kinds of stimuli were well distinguished by the patient. In relation to the rest of the body, the sensibility of the feet was, perhaps, less marked. The presence of a large amount of cicatricial tissue on the feet prevents, however, drawing any definite conclusions. The sense of localization seemed to be normal within physiological limits. The stereognostic sense and that of position of the members did not present anything abnormal.

SPECIFIC SENSES.—The visual power was reduced to one-third

of the normal, and the sense of chromatic perception was normal on both sides. The visual field and the eye ground was normal.

On both sides hearing equaled 0.5 with Galton's whistle. Perception of low, middle and high tones was good. Vibrations of the tuning-fork, when touching the cranium, were not perceived either on the right or on the left side.

Olfactory and gustatory sensibility was normal on both sides. Pleasant odors were readily distinguished from unpleasant ones. Tastes of various articles were readily and correctly recognized.

Speech was normal, save for some slowness of pronunciation and noticeable scanning. This did not interfere, however, with proper pronunciation of separate syllables. Single letters of the alphabet were properly pronounced. The patient's writing was legible, but tremulous. Besides, it was an effort for her to lead the penholder and after having written some lines she seemed to be exhausted (see figure 1).

PSYCHIC CONDITION.—The patient is able to read and to do such feminine work as her physical condition allows. The facial expression was gentle, although sad and stereotyped. Spontaneous as well as forced attention was good. Mental perception, within the limits of her intellect, was good. Memory was good. Although her general tone was sad, she entertained affective sentiments towards her parents. Her religious sentiment was highly developed and she spent long hours in prayer. The long years of suffering had not made her self-centred. She tried to cause as little trouble as possible to those about her. From choice, she preferred to avoid being noticed by strangers. She often spoke of her childhood companions, but without any bitterness of sentiment. While she did not entertain any hopes of recovery, she accepted with deep gratitude any kindness shown her. Her dreams were rather rich in action. She often dreamt that she was walking or dancing, but these pleasant impressions were marred by her dreaming of falling quite frequently. At times she had frightful dreams, imagining that people chased her. She ran on, however, without feeling tired or falling.

Case II—Robert X., seventeen years of age, Maria's brother. The patient had typhoid fever at three years of age. He was healthy up to the age of ten years. At this age he began to show some difficulty in walking. At first he felt shaky on his feet and then he began to fall. His sister's disturbance commenced at the same age. While these disturbances were developing, the patient also suffered from lancinating pains in the limbs. These pains progressively increased, and we were called in to examine the patient January, 1899.

The movements of the eye balls were normal, except for some occasional

horizontal nystagmiform movements when the eye balls were turned well outwards. The facial and lingual nerves were normal. Mastication and deglutition were normal. The muscular nutrition of the upper limbs was normal, and the active and passive movements of the limbs were normal. There were no disturbances whatsoever in these limbs.

The lower limbs presented nothing abnormal when in a horizontal position. Passive movements were performed without there being any resistance. Active movements of the thighs and legs were possible and well executed to the fullest extent. The patient could not hold the thigh flexed over the pelvis, however, while the knee joint was extended. When attempting these positions he repeatedly faltered in the act. The movements of the feet were quite limited. His gait was characterized by divergence of the legs and more than normal flexion of the thighs, but the heels were not retracted and the toes did not drag on the ground. At times there were decided oscillations of the whole trunk of the body, so that the gait resembled that of a drunkard. Besides, he was unable to hold himself on his feet without the help of an assistant. Unaided, he could hold himself on his feet only a few seconds, then fell helplessly. He could not succeed in bringing the tips of the fingers in apposition, even when his eyes were open. His inability to accomplish the act with his eyes closed was not much different in degree. He could not touch the knee of one limb with the heel of the other. The act of taking hold of some object, such as a glass, was accompanied by some tremor of the hand, but he carried the glass to the mouth without showing any tremor.

REFLEXES.—The pupils were equal and of medium size. They reacted well to light and accommodation. All the tendon reflexes of the upper and lower limbs were abolished. The cremasteric reflexes were also abolished. The plantar, epigastric and abdominal reflexes, on the contrary, were marked.

SENSIBILITY.—Tactile sensibility and that to pain was somewhat impaired in the legs and in the feet. This hypoaesthesia became less and less marked as the area examined was nearer the thighs. The reaction to cold was characterized in a similar manner.

SPECIAL SENSES.—Vision equaled 1 on both sides. The chromatic sense was normal, except that on the right side yellow was taken for red. The visual field was normal, as was the eye ground.

Hearing equaled 0.4 to 0.5 of Galton's whistle—on both sides. The vibrations of the tuning-fork touching the skull were not perceived on either side. Low, medium and high notes were correctly perceived.

Odor and taste were normal on both sides.

Speech was scanning to a certain degree.

Generally speaking, the disease gradually progressed during the period of 1899-1902. The hands were noticeably affected in 1900, as could be judged from the patient's handwriting. The parents, however, did not notice the trouble until 1901,—one year before the patient's death.

Io spero di guarire
Io non sono più tanto
malata come prima




FIG. 1. Handwriting of Maria X. in 1904.

Printed copy of writing: Io spero di guarire
Io non sono più tanto malata
come prima.

We examined the patient in July, 1902. We found that the reflexes and general sensibility were about the same as when we made our first examination—three years previously. Incoordination, however, was so marked that the patient could not take a step even when led by two attendants.

The urine contained a large amount of sugar (5.25%) and there was acetonuria. The patient died in diabetic coma.

AUTOPSY.—The autopsy was performed twenty-four hours after death, July 14, 1902. We were allowed to examine the cerebro-spinal system only.

The cranial bones were normal. The dura mater was normal, but the pia mater was somewhat opaque and slightly thickened, although easily detached from the convolutions. The lateral ventricles were normal. The basal ganglia were normal. The cerebellum did not show any appreciable diminution in volume. The spinal cord, on the contrary, was below the normal size. A transverse section showed a marked gray coloring of the areas corresponding to the posterior columns. This coloring was far more evident in the dorsal and lumbar regions than in the others.

(To be continued.)

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Original researches and other MSS. will be carefully considered, and if found unsuitable will be returned, if accompanied by stamped, self-addressed envelope.

DR. S. WEIR MITCHELL HONORED IN EUROPE.

We experience great gratification in announcing the election of Dr. S. Weir Mitchell, of Philadelphia, as *Membre Correspondant étranger of the Academie de Médecine*, of Paris. This election makes an epoch in the growth of friendly intercourse between the members of the medical profession in France and in the United States—a growth that has, for peculiar and deplorable reasons, not advanced as speedily as we should have wished.

Dr. Mitchell has earned for himself the distinction of being the second American to be honored by such an election, the first being Dr. Charles W. Stiles, of Washington, D. C., who was similarly honored in 1896.

Dr. Mitchell's election occurred on July 19, 1904. His membership is of the first division, in which the number of *Membres correspondants étrangers* is limited to twenty-five. This division comprises the following branches: anatomy, physiology, therapeutics, medical natural history, pathological anatomy, medical

pathology, hygiene and legal medicine. There is one election every year, two members being elected at a time.

Dr. Mitchell's name was first presented to the French Academy of Medicine this year. As a rule, candidates remain on the waiting list a long time—sometimes as long as ten or twelve years. Dr. Mitchell's election the very same year his name was presented is most gratifying. The proposition was made by the Physician of Ste. Anne—Dr. V. Magnan—who, although not personally acquainted with Dr. Mitchell, espoused his cause. Dr. Magnan displayed the energy of a young man in securing the necessary votes for the American candidate and felt quite jubilant over his success.

We extend our congratulations to Dr. Mitchell and trust that this new bond of friendship between the French and American medical professions will lead to still more intimate relations between the two nations.

XIVTH CONGRESS OF ALIENISTS AND NEUROLOGISTS OF FRANCE AND FRENCH SPEAKING COUNTRIES.

PAU, August 1-7, 1904.

1. ACUTE HEMORRHAGIC POLIENCEPHALITIS SUPERIOR. —BRISSAUD AND BRECY: The patient, a woman, thirty-six years of age, presented double ptosis, myosis and a peculiar condition of somnolence. On the ninth day after admission to the hospital the pulse was accelerated and the temperature high. Sudden death on the following day. Autopsy: inflammatory lesions with punctiform hemorrhages along the walls of the aqueduct of Sylvius, especially in the region of the nucleus of the Third pair, the corpora quadrigemina and the upper part of the Pons Varolii.

This case is similar to that reported by Gayet, in 1875, and those described since by Wernicke under the name of acute hemorrhagic polienccephalitis superior. The lesions are diffuse and predominate in the region of the aqueduct rather than presenting any systematized localization.

2. OPHTHALMIC MIGRAINE WITH TRANSITORY HEMIANOPSIA AND APHASIA. SUCCULENT HEMIFACE, PHOTOPHOBIA AND PALPEBRAL TICS.—

HENRY MEIGE: The patient, 73 years of age, is subject, since the onset of her menopause, to migraine with scintillating scotoma, cephalalgia, vertigo and nausea. The spells are accompanied by transitory hemianopsia with aphasia, paresis of the right face and tingling of the right arm. The spell is ended by a period of somnolence. After a series of these spells the right face presented slight paresis and was also edematous (succulent hemiface).

She suffers from involuntary winking that is due to tics. The starting point of the latter was migrainous photophobia. The tic persists independently of the spells and seems now to be of obsessional nature. The migraine phenomena may be ascribed to transitory vascular disturbances, the seat of which can only be surmised. An angiospasm of the ramifications of the Sylvian artery would explain the disturbances of speech, the facial paresis and the tingling of the arm. One must suppose a larger extension of the angiospasm, however, in order to explain the accompanying scotoma, hemianopsia and vertigo. The facial edema points towards the participation of the vaso-motor centres. There is probably a symptomatic trophedema in connection with the action of the sympathetic centres. There may also be a bulbar angiospasm,—as the migraine spells are accompanied by phenomena of anxiety.

Outside of the periods of the migraine spells, the patient's facial expression, attitude, speech and gait strikingly resemble the similar traits in progressive hemiplegia. The palpebral convulsions are not true convulsions, as they can be suspended voluntarily or by some accidental distraction of attention. Their onset and exaggeration is in direct relation to the photophobic preoccupations. This speaks in favor of tic. The tic is senile, however, in this case, and resembles more true spasms than does tic in the young. In the old there is an organic inferiority of the nervous centres and conductors.

3. ILLUSTRATED DOCUMENTS REPRESENTING ANCIENT SURGICAL TREATMENT OF PSYCHOSES.—

HENRY MEIGE presented about thirty artistic illustrations from the various European private and public art collections, especially Flemish and Dutch, representing cranial operations. Some of the illustrations represent simple blood-letting or applications of plasters for the relief of migraine, etc. Most of the illustrations, however, represent jugglers' practices known as *opération des*

pièrres de tête. It was believed in those days that derangement of the mind was caused by foreign bodies in the brain. Wasps, gad-flies and rats were among the imagined inhabitants of the brain, and in our own day some Europeans still speak of "spiders and beetles in the head." In the Netherlands insanity was spoken of in connection with *a stone in the head*. Chirurgical jugglers exploited especially this belief. They incised the cranial coverings of their patients and produced, during the operation, a stone, presumably extracted from the brain, but that in reality came from the hollow of the operator's hand where it had cleverly been concealed for the purpose.

In our own day many obsessional cases picturesquely paint sensations they experience from the presence of foreign bodies in the brain. Among the artists who painted the encouragement of these beliefs by Medicine, the excessive credulity and the odacious trickery were the following: Van Bosch, Van Hemessen, P. Bruegel, de Bry, Brouwer, Teniers, A. Both, N. Weydmans, Frans Hals and others.

4. CLINICAL AND THERAPEUTIC REMARKS ON SOME TICS IN INFANCY.—HENRY MEIGE AND FEINDEL: Precipitated speech is of frequent occurrence in the young subject to tics. They either sputter or have sudden interruption of speech. There is an undoubted relation between stuttering and tics. A similar mental status is at the root of both conditions. Similar principles of psychomotor discipline are applicable to these different psychomotor disturbances. Respiratory troubles met with in those subject to tics are primary and secondary. Some abrupt expiratory acts are due to tics of the upper limbs or the trunk of the body. Tics of beating or kicking one's self with the fists or feet are phenomena similar to the acts of scratching or gnawing automatically (onychophagia, cheilophagia). The starting point is an abnormal sensation that prompts these acts. By force of repetition the motor act becomes habitual even when there is no call for it. Such tics may, in their turn, start up local pains by repeated irritation. Although these new sensations are due to the motor tics, the patients generally believe the contrary. Applications of vaseline with quinine in it is, on account of its bitter taste, an excellent remedy for cheilophagia and onychophagia. Psychomotor discipline should be applied in such cases to the widest possible extent.

5. DELIRIOUS EUPHORIA OF THE CONSUMPTIVE. AN ANATOMOCLINICAL STUDY.—E. DUPRE: The symptoms were studied in their relation to the cellular pathology

of the brain. The patient was thirty-three years of age and suffered from sub-acute phthisis. There was fever, bilateral infiltration, formation of cavities in the lungs, laryngitis and rapidly progressing cachexia. During the entire course of the disease the patient, who was intelligent and cultured, presented classic euphoria, optimism, illusions, projects,—firmly hoping for the best and accepting without question any explanation for his pitiable condition. In conversation he smiled and laughed, looked radiant and bore an expression of beatitude. Many authors have commented upon this apparent radiance and the sad reality in such cases.

The patient died of asphyxic coma. At the autopsy was found: Caseous infiltration of the lungs. Fatty degeneration of the liver and kidneys. Enormous enlargements of the spleen and a small, flabby heart. The brain weighed 1.220 grams and there was no trace of meningo-encephalitic tuberculosis.

The cerebral fissures were slightly enlarged and the ventricles somewhat dilated. The pia mater was decidedly thickened in the region of the frontal lobes, although there were neither adhesions nor tubercular lesions. The thickening was in circumscribed patches and whitish.

Prof. Nissl made the histological examination. There was simple hyperplastic collogogenous meningitis without exudation, inflammation or specific characteristics. There was no diapedesis, slight proliferation of the vascular endothelium with disseminated yellow pigmentation around some of the endothelial cells. There were some cells, designated as *staebchenzellen*, in the immediate vicinity of the capillaries. Profound and diffuse lesions of the frontal nervous cells. Disappearance of the protoplasm, eccentricity of the nucleus, deformity of the nucleolus that was shrunken and its membrane wrinkled. Vacuolation of some of the pyramidal cells and hyaline degeneration of the majority of the capillaries. In the white substance: degeneration—commencing putrefaction. *Lacunæ*, dissolution of the medullary substance and some tracts of streptobacilli along some of the vessels. Similar although less marked lesions characterized the middle and posterior regions of the cortex. The pia mater was simply injected in this region. These lesions are of toxic nature and independent of vascular changes. The meningeal lesions are of more remote date and of different nature. The cellular lesions of frontal localization are the cause of the symptomatology. Tubercular subjects who do not present during life the mental symptomatology in question are free from the frontal lesions indicated. A similar de-

mental status is also found in cancerous subjects kept on heavy doses of morphine. The analogy between the demential conditions points towards intoxication as the cause of the euphoria in phthisical subjects. The intoxication is due to bacillus poisoning, hepatorenal insufficiency and subacute anoxhemia.*

TRANSLATIONS AND ABSTRACTS OF CURRENT LITERATURE.

THE EVOLUTION OF CONSCIOUSNESS TOGETHER WITH A DIAGRAM ILLUSTRATING CERTAIN HOMOLOGIES IN THE NERVOUS SYSTEM.—DR. W. H. B. STODDART: Consciousness depends on sensation. This fact was well illustrated in Strumpell's patient, whose only means of communication with the outside world was one eye; the other eye was blind, both ears deaf and the whole surface of the body anesthetic. Whenever the sound eye was closed, he went to sleep. In other words, he lost consciousness whenever the only means of communication with the outside world—his sound eye—was closed. Without sensation, the author remarks, consciousness cannot exist. The essential basis of consciousness is sensation. Sensation is probably experienced by every individual cell in the body. It is generally admitted that an ameba experiences sensation. It feels, it has sensation, it knows that it feels and is conscious. A thing that feels and does not know that it feels is inconceivable. Consciousness is not the only function of a unicellular organism. Its function is also that of digestion, excretion, movement, reproduction, etc. The observations of Carter, Romanes and others suggest that an ameba not only has sensation, but that it even possesses such highly developed mental attributes as reasoning and memory. The author concludes that every cell of the human body feels and knows that it feels, that it is a conscious unit and that it has a very elementary mind. The majority of the cells of the human body are represented in the central nervous

* We are indebted to Prof. Brissaud, of the *Revue Neurologique*, for the French proof sheets of this report.—Ed.

system. Huglings Jackson was the first to point out that the muscles of the body were first represented in the cells of the anterior horns of the spinal cord and in the nuclei of the cranial motor nerves; that the muscles were next represented in the Rolandic region of the cortex cerberi; that there was a further representation in the physical basis of consciousness, this being situated, in all probability, in the prefrontal lobes. To-day the mode of arrangement of the different parts of the nervous system is better understood, but the data presented remain advantageous in the study of the hierarchy of sensation.

There are some cells the sensation of which is not represented in the central nervous system, as is the case with the leucocytes and most of the cells of connective tissue. The representation of sensations of cells of the more highly organized tissues is explained by the author.

Fitness of reaction to a stimulus may be accepted as a criterion of consciousness. Fitness of reaction very frequently occurs, however, independently of the cerebral cortex. This is the case when an ordinary reflex action occurs. A classic example of this is found in the reaction to stimulus in a brainless frog. A fitness of reaction is also represented when the eyelid brushes away a foreign body from the conjunctiva, when sneezing results from irritation of the nostrils, coughing from irritation of the larynx, etc. In each case there is an attempt on the part of the organism to get rid of the offending body. Yet all these suitable reactions occur independently of the cerebral cortex.

The author concludes that there are many centers of consciousness, or colonies of conscious units, in the nervous system other than in the central cortex. Each of these colonies has a combined total consciousness of its own. These colonies, the author says, are severally the physical bases of the consciousness of separate individual parts of the organism, and are no part of the physical basis of consciousness of the organism as a whole. The consciousness of man is consciousness of no other part of his nervous system than his cerebral cortex, although there are many centers of local consciousness scattered throughout the organism (*Brain*, Part CIII, 1903).

HYPERTROPHY AND LESIONS (TUMOR) OF THE SUPERIOR CERVICAL SYMPATHETIC GANGLION IN A SO-CALLED CASE OF GENERAL PARALYSIS.—DR. D. DE BUCK: The case in question presented a fair picture of general paralysis, including the somatic and delirious manifesta-

tions. There were also pupillary signs. The disease seemed to be of a galloping type and the patient died shortly after admission to the hospital. The autopsy and a subsequent microscopic examination showed that the cerebral tissue was free from any lesion characteristic of general paralysis. The delirium was apparently due to auto-infection caused by polycystic degeneration of the right kidney, impairment of the left kidney and atrophy and sclerosis of the liver. The principal lesion, however, of interest in this case was a hypertrophy of the superior cervical ganglia. The right ganglion was about three times its normal volume and the left one was about the size of a pigeon's egg. This tumor was encased in a fibrous capsule. Microscopically, the noble elements of the ganglia were destroyed.

The pupillary symptoms, which the author attributed to the presence of general paralysis, were due, on the contrary, to the lesions of the cervical ganglia. The delirium was due to auto-intoxication. The principal affection, however, that was not diagnosed during life constitutes a clinical rarity. It was a fibro-sarcoma of the ganglion mentioned. The seat of such a tumor is particularly rare. Chipault is the only author who has reported a case of a myxomatous tumor of the superior cervical sympathetic ganglion. Neither Chipault nor the author of this article have found any similar cases reported in medical literature. The case did not present any epileptic manifestations except shortly before death.

Physiologically, it is difficult to draw any conclusions regarding the functions of the sympathetic system. While it is known to be vaso-motor and trophic in its function, extirpation of the sympathetic ganglia is possible in some instances, as in epilepsy, migraine, depressive psychoses, etc., without causing any serious or permanent vaso-motor or trophic disturbances. The author does not accept Bonnet and Poincaré's opinions that the primary lesions of the sympathetic were responsible for the symptomatic pathogenesis of general paralysis. While the clinical picture of the author's case simulated that of general paralysis, the matter did not go any farther, as the microscopic examination confirmed (*Bulletin de la Société de Médecine Mentale de Belgique*, February, 1904).

CONTRIBUTION TO THE STUDY OF THE INTERNAL ORGANS IN GENERAL PARALYSIS.—SERGE SOUK-HANOFF: General paralysis is characterized not only by profound alterations of the central nervous system, but also by that

of other nervous centres, such as the spinal cord, the spinal and sympathetic ganglia. The peripheral nervous system and the muscular system are also affected. In the midst of these various alterations it is sometimes difficult to point at the true source of the profound anesthesia of the internal organs. This anesthesia in the general paralytic may be expressed in various ways. Thus, for instance, a general paralytic may give birth to a child without experiencing any pain whatsoever. The author has seen a case of parturition in a general paralytic 22 years of age, without there having been any manifestation of pain whatsoever. The author says that such a complete absence of pain can be observed only in cases of profound narcosis. Dr. Semidaloff has seen two similar cases of parturition in the Alexeievsk Psychiatric Hospital, Moscow. Another case of general paralysis in which profound anesthesia was observed was one that suffered from a round ulcer of the stomach. The patient suffered from incoercible vomiting, wasted rapidly, and the disease was correctly diagnosed as being an ulcer of the stomach; but the patient did not complain of any pain whatsoever. The author attributes this absence of pain to profound anesthesia of the stomach in this case. In another case of general paralysis the autopsy showed extensive tubercular destruction of both lungs, but the patient never complained of any pain during life, nor did he show any signs of any bronchial irritation or cough. This profound anesthesia of the internal organs of the general paralytic must be due to the impairment of the various nervous systems. It is hardly justifiable to incriminate the demential condition of these patients as responsible agents in the abolition of pain. The author has observed cases of profound dementia with disturbances of consciousness that are sensitive to pain of all kinds. Among others, a microcephalic child, who is under the author's observation, is quite sensitive to various kinds of pain. The author concludes that the demential state of the general paralytic is not the essential factor in the abolition of the sensation of pain. A profound cellular alteration of the nervous system, as explained above, must be at the root of the trouble. Auto-intoxication may also be one of the factors in the impairment of the general sensibility (*Revue Neurologique*, April, 1904).

ON THE OCULAR SIGNS IN GENERAL PARALYSIS.—PROF. A. JOFFROY: The number of general paralytics examined was 227. Of this number 212 presented ocular disturbances. Inequality of the pupils was the first manifestation in 144 cases. Besides the alterations of the pupillary diameter,

the author found irregularity of the pupillary contour. This deformity occurred in 93 out of 125 cases. As a general rule, in general paralysis, the light reflexes diminish gradually, and finally disappear, while accommodation still persists. Accommodation is lost towards the end of the disease, when dementia is well established. The author differs with Ballet, who has formulated his opinion thus: general paralysis is characterized by "gradual and progressive internal ophthalmoplegia." The author disagrees with Ballet because, clinically, impaired accommodation is not the pre-eminent manifestation. The external muscular apparatus was impaired in 38 out of 227 cases. These lesions comprise only those caused by the affection in question. Among other lesions the author found ptosis in 12 cases, without other manifestations of impairment of the III-d pair. Paralysis or pareses of the muscles supplied by the III-d pair was found in 14 cases. Paralysis due to impairment of the IVth pair was found five times. Nystagmus was found five times. Spasmodic orbicular contraction was found twice. Sometimes these manifestations may take place after apoplectiform or epileptiform attacks; most frequently, however, these lesions do not depend on these attacks. When general paralysis was associated with ataxia, the pupillary manifestations were frequently ataxic in nature.

Hyperemia of the eye ground was frequently found. According to Keraval and Raviart this lesion is found in 42 out of 51 cases. Prof. Joffroy, on the contrary, found this lesion only in 27 out of the 227 cases, or in 12 per cent., as against 82 per cent. as indicated by the former authors.

The visual field was found normal in all the cases, and the author disagrees with Reznikow, who claims that this field is markedly impaired in general paralysis.

The author concludes that while the ocular signs are helpful in making a diagnosis in general paralysis, they are not pathognomonic of the disease (*Archives de Neurologie*, May, 1904).

GRAPHIC STUDY OF THE PLANTAR REFLEXES IN THE NORMAL STATE AND IN SOME SPASMODIC AFFECTIONS OF THE PYRAMIDAL SYSTEM.—DRS. HENRI VERGER AND JEAN ABADIE: The authors' conclusions are as follows:

1. In the normal state, a minimum excitation of the sole of the foot, by means of scratching or tickling, is followed by reaction both in the muscles of the toes, the leg and the thigh. This demonstrates that the plantar reflex may be considered as being com-

posed of a complexus of segmentary movements of the lower limb. This reflex is not caused by the reaction of certain muscles to the exclusion of others. The plantar reflex may be considered as being composed of three different constituents: planti-digital reflex, planti-tibial reflex and planti-crural reflex. Physiological research justifies this division.

2. The planti-digital reflex, properly speaking, presents two principal forms of about equal frequency during experimentation. One form is that of pure flexion and the other is that of flexion and consecutive extension. The normal extension is always of smaller amplitude than is the flexion that precedes it. This fact has up to the present escaped direct observation, but is revealed in graphic analysis. This explains why it has generally been affirmed that in the normal state the toe reflexes are expressed by flexion exclusively.

3. Variation of the intensity of the plantar excitation in no way changes the graphic form of these reflexes and particularly of the planti-digital reflexes.

4. In spasmodic affections of the pyramidal tracts a minimum excitation of the sole of the foot also provokes simultaneous muscular reaction in the different segments of the lower limb. A graphic study of the reflexes in these affections justifies a division of the reflexes similar to that enumerated above.

5. The most important and constant trait of the plantar reflexes in these affections is exaggeration. The excitable zone may often extend farther than the sole of the foot and involve the whole lower limb.

6. As regards the planti-digital reflex in particular in spasmodic affections of the pyramidal tracts, they may give the following graphic forms, regardless of the nature of the affection in question:

- a. The two normal forms.
- b. A form of consecutive flexion and extension of the great toe, the extension always being more ample and longer than the flexion. This is the more frequent form.
- c. A very rare form of pure extension, in which flexion is so abrupt that it is hardly registered, if at all.

This graphic study does not decrease the semiological value of Babinski's sign. The authors demonstrate, it is true, that extension of the great toe is not a pathological phenomenon due to alteration of the motor tracts. This extension may accompany flexion in the normal state. But in spasmodic affections of the

pyramidal tracts the amplitude and duration of this extension is so marked that the clinician is apt to take the phenomenon for a reversal of the normal manifestations—simply because in the normal state flexion alone generally manifests itself. In reality, however, there is very little difference between the normal and pathological reflexes. Indeed, graphically, extension often follows flexion in the normal condition. On the other hand, in pathological conditions, slight flexion may also precede extension. The difference consists in the following: in pathological conditions it is more marked and its long duration masks the flexion that precedes it (*Nouvelle Iconographie de la Salpêtrière*, January-February, 1904).

EPILEPSY: PATHOGENESIS AND THERAPEUTIC INDICATIONS. CONTRIBUTION TO THE STUDY OF THE PHYSIOLOGY OF THE THYROID BODY.—DR. AL. PARIS: The utmost importance is ascribed to the function of the thyroid gland. Hyperactivity of this gland is considered as an important cause in the pathogenesis of epilepsy. Hyperactivity of the glands of reproduction is placed as secondary in importance to that of the thyroid gland. Defective absorption of these secretions, due either to hypersecretion or insufficient elimination, results in epileptic manifestations. Abnormal accumulation of these secretions produces an exciting effect on the central nervous system. An epileptogenic constitution of the nervous system predisposes to this effect of the so-called internal secretions of the glands in question. From a clinical point of view, it is advisable to aim at moderation of the functional activity of the thyroid gland. In the treatment of epilepsy it is essential, the author claims, to moderate the functional activity of the thyroid gland and of the glands of reproduction. Accumulation of their secretions should be obviated. Accidental accumulation of toxins in the system is also apt to act as an excitant on the central nervous system.

Among some of the arguments adduced by the author in favor of the important rôle of the thyroid gland in the pathogenesis of epilepsy are the following: the severity of epileptic attacks is decreased with the decreases of the functional activity of the thyroid gland. The amelioration that takes place in epileptics subsequent to section of the cervical sympathetic nerve is considered as being significant. This nerve supplies some branches to the thyroid gland. In some cases of women the severity of the epileptic attacks is decreased during pregnancy. When the period

of reproduction is attenuated, the activity of the thyroid gland decreases and the severity of the epileptic attacks is also decreased. According to the author this is an important proof that the thyroid gland plays a most important rôle in the pathogenesis of epilepsy (*Archives de Neurologie*, March, 1904).

THE INFLUENCE OF BLINDNESS ON THE SPINAL, SENSORY AND MOTOR DISTURBANCES IN TABES.—

P. MARIE and A. LERI: The authors have tried to find out whether the occurrence of blindness in tabes prevented the development of the various disturbances common to the disease, whether the manifestation of blindness attenuated or helped disappear these disturbances when they were already in existence. The number of cases of tabes with blindness examined was forty-five. Of these only five were free from lancinating pains. All the others manifested, at various times, the characteristic lancinating pains. The pains were more moderate, however, than in ordinary cases without blindness. In 32 of these cases the disturbances appeared as follows: Sixteen cases manifested the disturbances after the visual trouble had set in. In two cases the visual and sensory disturbances set in at about the same time. In fourteen cases sensory disturbances manifested themselves before the blindness. From these observations the authors conclude that blindness does not prevent the onset of pains in tabes, as some authors believed. In some instances only did the sensory troubles take place synchronously, immediately preceding or following the onset of blindness. In the majority of cases these two phenomena take place at an interval of from one to four or more years. In some of the cases the lancinating pains set in eleven, fourteen and even twenty-six years after the visual disturbances. Therefore, there does not seem to exist any relation between the date of appearance of the pains and that of amaurosis.

The appearance of blindness does not seem to attenuate the existing disturbances. The pains are under no circumstances permanent and progressive. They are, on the contrary, intermittent and even transitory. In two-thirds of the cases pain is one of the first symptoms of tabes. In some instances the pain lasts for years, but it gradually diminishes in intensity and may finally disappear with the cause of the affection—the inflammatory process. The authors have found that in tabes with blindness the course of evolution of the pains is similar to that just mentioned. Four out of five patients, who have been suffering from pains for

various lengths of time, between 20, 18, 13 and 12 years, have had no relief after the onset of blindness. In one case the pains disappeared one year after the onset of blindness. In this patient the pains were rather slight in degree and had lasted for some five years. One case had suffered from pains for a period of some fourteen years, although he was blind twelve years of this period. He was free from pains only about one year before death.

The study of the relation of blindness to the motor disturbances has led the authors to conclusions similar to those given above. The onset of blindness does not immunize the patient either from sensory or from motor disturbances. Nor has blindness any attenuating effect on these disturbances. Amaurosis and the spinal symptoms of tabes seem to develop independently one from the other (*Revue Neurologique*, April 30, 1904).

CONTRIBUTION TO THE STUDY OF AUTO-INTOXICATION IN EPILEPSY.—DR. GUIDO GUIDI: Tabulated results of personal experiments and chemical analyses of the urine of many epileptic patients lead the author to the conclusion that epileptic attacks are in direct relation to the amount of ammonia salts excreted in the urine. A maximum amount of ammonia salts in the urine precedes, accompanies or follows an epileptic attack. Some tables show the augmented amount of ammonia salts during the day of the attack. The maximum amount of the ammonia salts in the urine does not necessarily take place on the day of the attack. Most frequently this maximum amount is found on the day following the attack. The author explains the fact by saying that during the attack there is an attempt on the part of nature to liberate the system from the noxious effects of the abnormal amount of ammonia salts. The attack itself is, in a way, the safety valve, and a large amount of the salts is expelled. During the attack, however, the muscular activity tends to increase the amount of the noxious elements and the latter are, consequently, expelled on the day following the attack. According to the author, the augmented amount of ammonia salts is due to marked acidity of the blood, which, in its turn, is the cause of the epileptic fits. The compounds of the elements in question act on the epileptogenic centres and produce the attacks. The augmented amount of ammonia salts in the urine on the day following the attack expresses the attempt of nature to liberate the system from the noxious effects of the salts mentioned. In order to assure himself that his conclusions were correct, the author experimented on two groups of epileptics, one having been kept on a

milk and the other on a mixed diet. In both instances he found that the cause of the attacks was the augmented amount of ammonia salts. In eighty examinations of these cases he found that there was always an augmented amount of ammonia salts after the attacks. According to the author there is a cyclic increase and decrease of the ammonia salts in the blood. The maximum increase of these salts in the blood is expressed by an attack. Then follows excretion of the noxious elements. Progressive accumulation of the same is then resumed as has been explained (*Annali dell'Institute Psichiatrico della R. Universita di Roma*, Vol. II, 1902-1903).

CONTRIBUTION TO THE ANATOMICAL STUDY OF THE POSTERIOR COLUMNS OF THE SPINAL CORD.—

J. NAGEOTTE: The endogenous fibres of the posterior columns of the lumbar region should be divided into two classes: fine and coarse endogenous fibres.

The coarse endogenous fibres furnish: a bundle of fibres in the cornu-commissural zone and in the median sacral triangle. The latter is also known as the triangle of Gombault and Philippe. This bundle of fibres is the lower extremity of the descending medio-peripheral bundle. The ascending course of the latter constitutes, in the dorsal region, the column of Hoche.

The median triangle is entirely distinct from the centrum ovale of Flechsig, that is, a radicular column.

Some of the endogenous fibres are horizontal, while others are perpendicular. The perpendicular fibres are scattered all along the column of Burdach. Some of them can be found in the column of Goll—in the cervical region.

The zone of Lissauer is made up of fine vertical endogenous fibres that are quite condensed in this region. They are not of radicular nature, as is generally thought. They degenerate at a late period in tabes.

The network of the fine fibres of the posterior horns is of endogenous nature.

The column of Clarke does not seem to receive any fibres from the posterior roots situated below the 3-d lumbar root.

The *bandelette externe* does not touch the posterior horn at any point, assuming a complicated form in the lower dorsal region that represents an M-shaped figure on each side.

The fibres of the *bandelette externe* are radicular fibres of medium length in the entire intra-medullary region; those of the lumbo-sacral region do not reach the column of Goll.

The long radicular fibres pass through the postero-external field, but not through the *bandelette externe*.

The marginal zone of Westphal, or anterior radicular zone, is composed, besides the endogenous fibres, of short radicular fibres only (*Nouvelle Iconographie de la Salpêtrière*, January-February, 1904).

NOTE ON THE HYPOTONIC FLATTENING OF THE FEET IN GENERAL PARALYSIS.—CH. FERE: Flattening of the foot in the erect posture is more marked in the general paralytic than in the normal subject. Muscular hypotonia in the general paralytic is responsible for this fact. The author found muscular hypotonia in 11 out of 28 general paralytics, or in 39.28 per cent. In another group of 62 general paralytics 28, or 54.16 per cent. of the cases, presented muscular hypotonia. The difference of the length and breadth of the foot in the respective postures is presented in photographic figures. It was difficult to make a systematic study of the progressive course of the muscular hypotonia because most of the general paralytics increase in weight after admission to the hospital. On the other hand, flattening of the foot is masked when wasting of the body coincides with the evolution of hypotonia. Nevertheless, it is possible to observe a considerable flattening of the foot in the latter instance. This coincidence was observed in 4 out of the 28 cases of muscular hypotonia mentioned in the group of 62 cases of general paralysis.

Muscular hypotonia causes a diminished height when the subject is in an erect posture. In a series of three groups of general paralytics, of 31, 29 and 34 cases, respectively, the average difference in height during the erect and recumbent postures was two centimeters in favor of the recumbent posture. In 11 subjects this difference was over three centimeters. Impaired voluntary extension of the general paralytics accounts for the diminished height in the erect posture. Thus, in 165 epileptics, for instance, the difference between the lengths of the body is only one centimeter in favor of the recumbent posture. The preceding explains the following fact: the length of the body is increased after death from 1 to 2 centimeters as compared with the measure taken during life in the recumbent position. In general paralytics presenting hypotonia, on the contrary, there is no change in the length of the body after death. The maximum elongation may attain some few millimeters (*Nouvelle Iconographie de la Salpêtrière*, January-February, 1904).

ON THE PRODUCTION OF SLEEP, GENERAL AND LOCAL ANESTHESIA BY MEANS OF ELECTRIC CURRENT.—PROF. STEPHANE LEDUC. Sleep can be experimentally induced by using a continuous electric current. The interior resistance should be feeble, permitting of gradual increase of the electromotor force in the circuit (accumulators or coils with a collector, reductor, etc.). With these currents the function of the central cerebral centres can be instantly inhibited, without causing pain and leaving the respiratory and cardiac centres intact. The induced sleep is calm in nature, can be prolonged as desired and there is complete cerebral anesthesia. The somniferous condition can be prolonged or suspended by continuing or suspending the electric current. The induced sleep is not followed by any after effects.

On opening the current, the animal shows some contractions and contractures, but it does not seem to suffer any pain. If the current is induced slowly, in order to avoid the stage of contraction, there is a stage of excitation analogous to that observed during chloroforming. Under these conditions it takes a longer time to induce sleep.

At the opening of the current there is generally evacuation of the bladder and the bowels.

The preferable currents are those of from 150 to 200 intermitances per second, passing in the minimum possible time, at a tension of from 12 to 30 volts, without self-induction in the circuit, marking from 2 to 10 milliamperes, according to the animal and the cathode applied to the head.

In man, local anesthesia can be induced with the same current. In this instance the cathode should be placed along the course of a sensory or of a mixed superficial nerve, as the Ulnar nerve at the wrist, for instance. With a current of certain intensity may be caused complete anesthesia of the areas supplied by this nerve. A certain degree of tingling may accompany the anesthesia (*Annales d'Electrobiologie, Tome, V.*).

ON SUBCUTANEOUS INJECTIONS OF SALINE SOLUTION IN VARIOUS PSYCHOSES.—DR. B. S. GREIDENBERG: The author experimented on 36 cases, 20 of which were women. The forms of the psychoses were principally of maniacal nature. Eight of the entire number comprised cases of melancholia, katatonia, general paralysis, primary dementia and acute febrile asthenia. A normal saline solution was used and every patient had had some fifteen injections. The injections were

given in increasing doses, beginning with 250 c.c. and ending with 1500 c.c. From these experiments the author draws the following conclusions:

1. Serotherapy seems to be indicated in the treatment of psychoses.

2. The subcutaneous method seems to be the most preferable one.

3. The action of these injections is sedative. Psychomotor excitation is decreased, the appetite is increased and the desire for sleep is restored.

4. In many instances the action of the injection supplants that of a prolonged warm bath.

5. Saline solutions are indicated principally in cases of acute infectious conditions and in auto-intoxication. The particular indications for injections in such conditions are: psychomotor excitation, insomnia and loss of appetite.

6. The patients readily submit to the operation and the after effects are nil. Antisepsis should be observed in performing the operation (*Report, IXth Medical Congress, Russia, St. Petersburg, 1904*).

IN REGARD TO RAYNAUD'S SYNDROME.—DR. PAUL MASOIN: This syndrome is found particularly in conditions of inertia, in the idiotic and in the epileptic. Among 425 of his cases at the Gheel Colony, the author found 65 epileptics and 150 idiots and imbeciles who were subject to this syndrome. The syndrome is mostly found in cases of epilepsy, idiocy and imbecility that are markedly degraded from a physiological point of view. The dystrophic alterations may, in some instances, be caused by peripheral neurites, but in the majority of cases the neuritis is rather the consequence than the cause of dystrophic disturbances. Besides, angio-neurotic troubles may take place without there being any neuritis. This is seen in hysterical cases. Similar phenomena are seen in advanced cardiac cases. In the latter instance it is obvious that the local circulatory disturbances are the causes of the trouble. Similarly, in hysterical subjects the stasis is of spasmodic origin. In the idiotic, epileptic and debilitated the cause may be local, such as cold, etc., or due to some central lesion with peripheral degeneration. In other cases, again, the cause may be of vascular origin properly speaking (endarteritis). All these causes may be more or less combined in one subject. The author suggests, therefore, that the denomination Raynaud's disease

might be exchanged for a more appropriate term—Raynaud's syndrome. This term, the author says, seems to be more indicative of the condition in question (*Bulletin de la Société de Médecine Mentale de Belgique*, February, 1904).

ON THE USE OF CAUTION CARDS IN ASYLUMS.—J. MARMAN: Labeling and segregating of suicidal and homicidal patients has its advantages as well as disadvantages. From one point of view inspection of such patients is far easier when each is labeled by a caution card, because the patients are segregated. The grouping of such patients together renders a less number of attendants necessary. This is important because it is easy under such circumstances to place a responsible attendant in charge. Finally, the focalizing and grouping of this class of cases is a guarantee that they will individually receive special attention. Some of the disadvantages are: under these conditions it is generally necessary to group together inharmonious cases. The effect is often depressing and deteriorating to some of these patients. The group of caution-card cases is quite apt to lead the physician and attendants into a routine way of handling them. Last, but not least, is the fact that under these conditions there is an unfortunate tendency to prolong as much as possible the period of special supervision. While it is easy to have the attendant sign the caution-card and to thus force on him or on her the responsibility of the case, it is far more difficult to know when to remove the responsibility. As regards the patients, prolonged segregation of such patients is more suggestive of suicide than of recovery. It is most important that the strain of constant supervision should be relaxed at the earliest opportunity. With this end in view the suicidal cards should be regularly inspected. The attendant in charge should be frequently consulted as to the general conduct of the patient. Any mental improvement, however slight, should be noted. The Commissioners in Lunacy insist on a frequent revision of the list of these patients, and, when properly possible, on the reduction of the number of these subjects (*The Journal of Mental Science*, April, 1904).

THE PRODROMATA OF PSYCHOSES AND THEIR MEANING. — DR. T. S. CLOUSTON: An attack of mental disease is generally not a simple or localized phenomenon. The study of mental attacks indicates that there is solidarity of action of the whole brain and of the whole of the nerve centres in the cord and the special ganglia of the organic system of the body. General considerations seem to point to the fact that the lower parts

of the sensory apparatus very often break down before the mental apparatus in the highest regions. They seem to prove the mental cortex to be the centre of the organism and teleologically its end. In this way they point to a greater resistiveness against disease in the higher centres. They show that it is chiefly in the brains hereditarily predisposed to the psychoses, or those whose defence is weak, that this natural resistiveness breaks down. In subjects not so predisposed all the symptoms that commonly constitute the prodromata of insanity may run their course without being followed by an attack of insanity. These considerations point strongly to the importance of a more careful study and attention to such preliminary symptoms in predisposed persons. They emphasize the view that the whole class of mental diseases should be regarded and treated not as local disturbances, but as widespread departures from the normal physiological condition of the whole organism (*Journal of Mental Science*, April, 1904).

MEDICO-LEGAL ASPECT OF POST-TRAUMATIC MENTAL TROUBLES.—DRS. MARIE AND VIOLET: The authors had studied one hundred cases of cranial traumatism followed by mental disturbances. The medico-legal suggestions made are rational and most applicable to the existing municipal conditions in France.

Discussing this paper, Dr. Vigouroux said that it was difficult to establish the pathogenic rôle of traumatism in the genesis of dementia and the insanities. This difficulty was encountered even when an autopsy could be performed. He cited a case of a general paralytic of classic form, in whose frontal lobe he had found a bullet. The latter was adherent to the internal table of the cranium and had perforated the dura and pia mater and was partly imbedded in the cerebral tissue. There was no inflammatory process around the bullet, while the diffuse meningo-encephalitic lesions were classic. The bullet had been lodging in the brain five years. He questioned whether it was possible to affirm that the bullet had played any rôle in the genesis of the disease, and if so, what the rôle was. In another case, one of senile melancholia, he found three bullets in one parietal lobe,—without there being any signs of reaction. A third case sustained a fall and developed general paralysis a few months later. This patient was syphilitic, an alcoholic and had had lead poisoning. The autopsy did not reveal any traumatic lesions. It is possible that the fall was the consequence of the general paralysis.

Dr. Briand said that the question was a complex one because

traumatism was not always followed by psychoses. In fact, the contrary was not uncommon. He cited a case of hypochondria with suicidal tendencies. This patient had lodged a bullet in his brain and the bullet was left to remain there. The patient became free from his hypochondriacal preoccupations.

Dr. Marie said that there was no doubt that in some instances traumatism was the cause of the onset of psychoses (*Archives Général de Médecine*, May 24, 1904).

ON NEURONOPHAGIA. SOME NORMAL AND PATHOLOGICAL RELATIONS BETWEEN NERVOUS AND NON-NERVOUS ELEMENTS. A CRITICAL AND EXPERIMENTAL STUDY. — DR. UGO GERLETTI: According to some authors, nervous cells, while undergoing pathological processes, may be destroyed by cellular elements that surround them. This phenomenon is generally called neuronophagia. There are many different opinions regarding the nature of the elements destined to enact the phagocytosis. The author disagrees with some of the views regarding the subject. Primarily, he objects to the opinion that the round nuclei found around the nervous cells under normal conditions are white blood corpuscles. These cells are, according to him, neuroglia nuclei. He also objects to the opinions of some authors regarding the possibility of migratory connective tissue cells infiltrating nervous tissues, unless it be in cases of destruction of all their elements, including the adventitia of the vessels. Under these circumstances phagocytosis is enacted by the migratory connective tissue cells. Then, however, the process is simply that of phagocytosis and applies to all the tissues in question. With these specifications in hand, the term neuronophagia remains applicable only when the process involves nervous cells exclusively. According to the views here criticized, the neuroglia enacts this particular phagocytosis. The author then tries to find whether this condition exists in reality. With this point in view he has made researches in normal histology, experimental pathology of the nervous cells and, finally, has studied various affections of the nervous cells in man. He concludes that in normal tissues it is quite common to find several neuroglia cells around the nervous cells and in the retraction cavities of the nervous cells themselves. He then considers Marinesco's neuronophagia theory unfounded. The study of the senile nervous centres also leads the author to disapprove of Metchnikoff's opinion that the senile nervous centres are the seats of intense neuronophagia (*Annali dell'Istituto Psichiatrico della R. Università di Roma*, Vol. II., 1902-1903).

ON FAMILY PSYCHOSES.—PROF. DOMENICO VENTRA: It is hardly justifiable to speak of family psychoses in the sense of similarity of the form of the psychoses, time of onset, etc., in different members of the same family. Among the psychoses generally spoken of as familial psychoses are insanities in twin children, suicides and idiocy in the same family. The first hardly corresponds to familial psychoses. Suicide cannot be elevated to the dignity of an entity as a psychosis; and idiocy may be called familial idiocy only when it is of the amaurotic form. The so-called familial psychoses may be classed as follows in order of frequency of occurrence: mani-depressive conditions, dementia precox, progressive general paralysis, melancholia, paranoia and degenerative insanity. Psychoses with imbecility also enter into this class of cases. It is well to note, however, that familial psychoses do not exist. It is more rational to speak of psychoses that occur in members of the same family (*Il Manicomio, Arch. di Psichiatria e Scien. Affini*, No. 1, 194).

COCAINE PSYCHOSIS.—PROF. DOMENICO VENTRA: Toxic psychoses form a special group of affections in psychiatry. These psychoses are characteristic of themselves and vary only with the individual characteristics. Cocaine psychosis, properly speaking, is rather a rare manifestation. Most frequently one meets with morpho-cocaine psychosis. This form of psychosis is far more difficult to cure than is the pure cocaine psychosis. This is due to the fact that in morpho-cocaine psychoses the poisoning of the system is complex. An example of this may be seen in cases of morphinization that is preceded by chloroformization. Cocaine acts principally by altering the nutrition of the nervous cell. The psychic, sensory and intellectual disturbances are due to these nutritive disturbances. It is possible to obtain complete recoveries in cases of cocaine psychoses. Recidivism is far more rare in cocaine psychoses than it is in morphinism. Cocainism is due, in the majority of cases, to indiscriminate administration of cocaine in cases of morphinism (*Il Manicomio, Arch. di Psichiatria e Scien. Affini*, No. 1, 1904).

PATHOGENESIS OF MUTISM IN PRIMARY DEMENTIA.—DR. M. L. BIANCHINI: Mutism is met with in 30 per cent. of the bulk of the cases of primary dementia and in 50 per cent. of the complex cases of hebephrenic and katatonic dementias. Mutism is essentially characteristic of katatonia (70%) and of hebephrenia (60%). Mutism is not characteristic of paranoidal dementia. Mutism is five times more frequent in

males than in females. This simply because woman is more talkative even during pathological conditions. Aboulia is at the root of the manifestation of mutism. In other words, there is an impairment of voluntary synthesis due to a pathological condition of the higher psychic centres. The motor centre of speech remains intact. Mutism properly belongs to the group of cases of negativism. There is submersion of the psychic faculty of speech (*Il Manicomio, Arch. di Psichiatria e Scien. Affini*, No. 1, 1904).

HEREDITARY SPASTIC PARAPLEGIA. ITS RELATION TO FRIEDREICH'S DISEASE AND ITS CLAIM TO BE CONSIDERED AS A CLINICAL ENTITY.—DR. H. CAMPBELL THOMSON: Summing up his remarks, the author says: The disease is characterized by premature degeneration of the lateral tracts with the posterior, and possibly other columns occasionally participate to some extent. The disease has a tendency to run in families and apparently, although to a less extent, to be actually hereditary. Isolated cases are also frequently met with. There is a very close relationship between it and Friedreich's disease. It is distinguished from the latter, however, by the predominance of the spastic symptoms. Here and there, however, as might be expected, the two diseases merge one into the other (*Brain*, Part CIII, 1903).

DELINQUENCY AMONG THE SARDINIAN INSANE.—DR. SANNA SALARIS: From a study of 62 cases of insanity with manifestations of violence the author draws the following conclusions:

1. While different forms of mental disease may be accompanied by similar modes of reaction, this mode of reaction is far more marked in the epileptic and in the paranoiac.
 2. Race has a marked influence on the form and degree of violent reaction. This influence is also observed among the insane. Insane of countries in which criminality is prevalent are also apt to manifest a marked degree of violent reaction.
 3. Stigmata of degeneracy are at a minimum in the paranoiac and the general paralytic as compared with the epileptic and neurasthenic.
 4. Paranoid or simply superstitious ideas are frequently met with among the insane of Sardinia. The combination of these ideas is the main basis of their delinquency and criminal acts (*Archiv. di Psichiatria*, Vol. XXV, Fasc. I).
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PATHOLOGICAL ANATOMY OF COMBINED TABETIC SCLEROSES.—O. CROUZON: Typical cases of tabes present other lesions than simply sclerosis of the posterior columns. Besides the lesions of the posterior columns one finds, in one out of ten or fifteen cases, lesions of the lateral columns. The term combined tabes is applied to cases presenting both lesions mentioned.

Combined tabetic sclerosis is expressed clinically by a symptomatic triad: tabetic gait with dragging of the limbs, paraplegia and extension of the toes.

When the lesions of the posterior and lateral columns are limited to a system of fibres they are called systematic. When the lesions are irregular, depending on vascular, meningeal and lymphatic lesions, the combined sclerosis is designated by the term pseudo-systematic combined sclerosis.

The author relates seven cases, five of which he explains by the lymphatic theory. Two of the cases are doubtful and may be due to lesions of Clarke's column. It is probable, however, that they were also caused by meningo-lymphatic lesions.

While combined tabetic sclerosis may be of systematic nature, it is more frequently of pseudo-systematic nature. This sclerosis cannot be sufficiently explained without the presence of meningeal and lymphatic lesions.

The nature of the anatomical process of these scleroses is similar to that of tabes and the pseudo-systematic distribution of their lesions argues in favor of the theory opposing the constant systematization of lesions in tabes (*Nouvelle Iconographie de la Salpêtrière*, January-February, 1904).

RAYNAUD'S DISEASE AND MULTIPLE CUTANEOUS NEUROTIC GANGRENE IN AN IDIOT—DR. D. DE BUCK: The seats of the gangrenous patches did not follow the courses of any particular nerves. The patient, an idiot, succumbed to the affection. The profound idiocy of the patient precludes the idea of there having been any hysterical element in the pathogenesis of the affection. Besides, marked central and peripheral lesions were found under the microscope. Cellular, vascular and perivascular lesions were found particularly in the regions around the central canal of the spinal cord,—between the anterior and posterior horns. It is generally agreed that the sympathetic nervous fibres have their origin in that locality. The peripheral lesions may possibly have been due to extension of the primary central lesions. The author is of opinion that the seat of the lesion that causes the peripheral vaso-motor disturbances consid-

ered here as Raynaud's disease and multiple cutaneous neurotic gangrene is in the peri-ependymal central gray substance of the spinal cord. In the author's case, some accidental irritation, added to the defective state of the nervous system of the subject, brought about the vascular lesions with the consequent sclerosis and gangrene (*Bulletin de la Société de Médecine Mentale de Belgique*, February, 1904).

THE SIGNIFICANCE OF LUMBAR PUNCTURE IN PSYCHIATRY. —PROF. NISSL: The author reports 166 personal cases in which lumbar puncture was practiced. Six of these cases comprise normal individuals. The amount of liquid drawn was from 3 to 5 c.c. Cases of melancholia and mani-depressive conditions were not operated upon. The conclusions are rather conservative, and a résumé is given of most of the works already published by the French and other authors. The author considers lumbar puncture as an operation and advises not to use it indiscriminately. In the six normal individuals he observed some untoward effects. The after effect takes place not immediately after the puncture, but some five or more hours later. In some instances the subjects suffered from nausea, vomiting, headache and general malaise. One of the normal subjects, a physician, suffered from after effects during a period of some ten days. Rest in bed in a supine position, after the operation, prevents the onset of the untoward effects (*Centralblatt fuer Nervenheilkunde und Psychiatrie*, No. 171, Vol. XXVII).

PARKINSON'S DISEASE.—PROF. F. RAYMOND: Parkinson's disease is generally apt to attack robust subjects free from hereditary taint. The tendon reflexes are found now decreased, now normal, now exaggerated. Exaggeration of the reflexes is most frequently present, even when the muscular atrophy is pronounced. Voluntary muscular movements are impaired, tremor is marked and deformity is particularly marked around the articular regions. Muscular atrophy and rigidity is particularly characteristic. Psychic impairment is not the predominating feature. In some cases mental depression and irritability may be noticeable. Towards the end of the disease deglutition becomes difficult and the voice impaired. Death is generally caused by an intercurrent disease. The causation of the disease is still obscure. Moral shock, fright, traumatism, etc., are considered as being some of the causes. The majority of the cases have a sudden onset. Disease of the thyroid gland has been incriminated by some

authors as being the cause of the disease, but this theory needs further confirmation (*Nouvelle Iconographie de la Salpêtrière*, January-February, 1904).

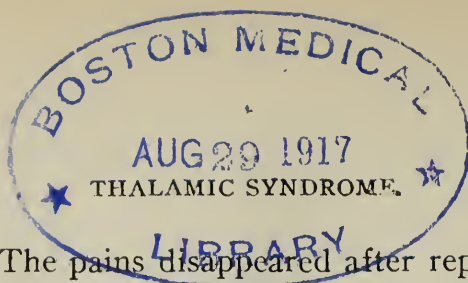
(*From Archives Générales de Médecine*, May 17, 1904):

1. PRIMARY CEREBRAL ACTINOMYCOSIS.—DRS. ENRIQUEZ AND SICARD report this case. It was impossible to find how the parasite had gained access to the brain, and the authors think that theirs is the first case of primary cerebral actinomycosis in France. An examination of the cerebro-spinal fluid during life indicated the presence of a cerebral tumor.

2. A CASE OF ACUTE MENINGITIS DURING THE COURSE OF SECONDARY SYPHILIS; DEATH REGARDLESS OF ANTISYPHILITIC TREATMENT.—DRS. SICARD AND ROUSSY: The meningitis developed seven months after the appearance of a chancre. Terminal hemiplegia characterized the disease. The case was one of acute cerebro-spinal meningitis of syphilitic nature. Histologically, there was generalized embryonic infiltration of the soft meninges and the vasculo-conjunctive tissues of the brain and the spinal cord. Secondly there was a partial thrombosis of the Sylvian artery. Cytologically, there was ample indication that the process was of an acute nature. Therapeutically, medium doses of mercury were given without any results whatever. This shows that in secondary syphilitic infection the treatment should be far more energetic.

3. NEURALGIA OF THE TRIGEMINAL TREATED WITH INJECTIONS OF COCAINE IN "LOCO DOLENTI."—DRS. BRISSAUD AND H. GRENET: In 1900, the patient underwent resection of the inferior maxillary nerve. After the operation there was amelioration during a period of several months. The pains then reappeared and injections of cocaine were then made in the painful points. The dose used was one centigram. This treatment was followed by relief for a period of one year. The cocaine treatment seems to be preferable to surgical intervention. It is worthy of note that regardless of the resection of a part of the inferior maxillary nerve the sensibility of the parts that it supplies is being re-established.

Dr. Brissaud remarked that it is difficult to explain the restitution of sensibility after resection of a considerable part of the inferior maxillary nerve. He has also seen restitution of sensibility in a case in which a considerable part of the superior maxillary nerve had been resected. The neuralgic pains also reappeared at



the same time. The pains disappeared after repeated injection of cocaine. This proves the excellency of Pitres' method.

4. THALAMIC SYNDROME.—DRS. THOMAS AND CHIRAY presented a case which, they thought, had an isolated lesion of the optic thalamus. They thought their case was similar to the two others presented by Déjerine and Egger. They thought it was justifiable to speak of a thalamic syndrome. The characteristics of this syndrome are as follows:

Slight hemiplegia contrasting with the marked, persistent and permanent disturbances of sensibility.

2. Appearance of chorataxia and athetosis in the hemiplegic area following the regression of the hemiplegia.

3. Marked disturbances of sensibility in the affected area. From the very beginning intense central pains are present on the hemiplegic side. Objective hemianesthesia of the skin is most evident as regards touch, pain and temperature. There is also marked dysesthesia, error of localization, and error of the quality of a given sensation. Deep sensibility is far more impaired than is superficial sensation. The patient loses completely the sense of stereognostic sensation, and the sense of perception of attitude.

4. In all cases there is absence of Babinski's sign, regardless of the presence of exaggerated reflexes.

5. Dr. Déjerine insisted on having seen in these cases toe reflexes when the toes were flexed. In some of his cases the diagnosis was confirmed by autopsies. Dr. Raymond agreed with Dr. Déjerine on this point.

Dr. P. Marie remarked that the atheto-choreic syndrome is the rule in hemiplegias of infancy involving the optic thalamus. Yet the syndrome is rare in adult or in senile hemiplegias with similar anatomical localization.

6. A CASE OF SUCCULENT FACE.—DR. P. BONNIER presented a case in which the lesion was neither cerebral nor spinal, but exclusively bulbar. He remarked that at a preceding meeting Prof. Raymond and Dr. Courtellemont presented an hemiplegiac with a succulent hand subject to congestive spells. Dr. Dufour, on the contrary, spoke of the edematous hand due to traumatic spinal lesion. Hence, a lesion of the same functional system situated at different heights may produce identical manifestations.

7. HEPATIC STEATOSIS IN THE INSANE.—DR. MAURICE DIDE: Hepatic steatosis is quite frequent among the insane. It may be said where steatosis is found the function of about two-thirds of the hepatic cells is impaired. Hepatic

steatosis is met with especially in cases of mental confusion (Ballet) and of katatonic dementia (Dide).

8. OCULAR DISTURBANCES IN DEMENTIA PRECOX.—DRS. DIDE AND ASSICOT: In 30 out of 50 cases the eye ground was of a peculiar grayish condition. This "washed out" condition of the papillæ is due to the congestive spells and to insufficient irrigation. This condition of the eye ground is sufficiently frequent of occurrence to constitute an objective symptom of the mental form.

9. ACUTE ANTERIOR POLIOMYELITIS OF AN ADULT WITH FOCAL LESIONS.—DRS. A. LERI AND A. K. WILSON: The patient was 30 years of age and the paralysis dated since the age of 23 years. The onset of the disease was sudden and was accompanied by fever. The clinical manifestations were those of acute ascending paralysis. There was no marked trouble of disturbance of sensibility. When the acute symptoms disappeared everything pointed towards acute ascending paralysis probably of polyneuritic origin.

The autopsy revealed complete, almost symmetrical, destruction of the greater part of both anterior horns of the spinal cord, in the cervical enlargement, between the sixth and eighth segments, on the one hand, and in the lower and middle lumbar regions on the other. These focuses had the aspect exactly like those found in old infantile paralysis. The vessels in the centres of these focuses were altered. This seems to point to a vascular origin of the lesion. The nerves presented decided interstitial neuritis, but this was of secondary nature. The degeneration of the muscles was similar to that which follows section of nerves or spinal lesions. According to the authors, this case demonstrates the possibility of the existence of focal lesions in the anterior horns of the adult, the lesions being absolutely similar to those found in infantile paralysis. Van Gehuchten presented a similar case at the recent Congress held at Brussels.

10. FAMILIAL CEREBELLAR ATROPHY.—DRS. BOURNEVILLE AND CROUZON: Two brothers, idiots, presented similar symptoms of spasmodic diplegia. In both cases the autopsy revealed cerebellar atrophy. This fact seems to indicate the anatomical substratum of a form of familial spasmodic diplegia.

11. BLINDNESS AND THE PROGNOSIS OF TABES.—DR. E. TERRIEN: Blindness was one of the early symptoms of the disease, but the incoordination developed rapidly and progressively until the patient had to be confined to bed. Consequent-

ly, tabetic amaurosis has no attenuating influence on motor inco-ordination.

CONGENITAL NERVOUS AFFECTIONS.—DRS. CHAR-
RIN AND LERI: Hemorrhages of the spinal cord and of the other organs are quite frequent in fetuses aborted by sick mothers. The abortions are not caused by these hemorrhages, but the latter are the causes of many pathological conditions when the pregnancies go on to full terms and the children are brought into the world. These hemorrhages may destroy the fibres of the nervous system already formed, and may be the starting point for the formation of cavities and scleroses of the nervous system. These hemorrhages depend on the disease of the mother or of the fetus, but not on difficulties during childbirth. The author has observed eight cases in question that presented lesions of the cerebro-spinal axis. Hemorrhages in the Rolandic region were found in three of the cases (*Progrès Médical*, May 21, 1904).

SCLEROSE EN PLAQUES. CONTRIBUTION TO THE STUDY OF THE PATHOGENESIS OF HEREDITARY SPASMODIC PARAPLEGIA.—PROF. R. MASSALONGO: Heredity alone is not necessarily the sole factor in the causation of the disease. Heredity predisposes the subject to the affection, but many other conditions may contribute to the onset and development of the affection. Some morbid conditions during intra- as well as during extra-uterine life may be the exciting causes of the manifestation of the disease in one or more members of the same family. Acquired affections are most frequently the causes of the manifestations of hereditary ataxia (*Rivista Critica di Clinica Medica*, April, 1904).

ON THE CENTRIFUGAL FUNCTION OF THE POSTERIOR ROOTS OF THE SPINAL CORD.—DR. C. POLI:
1. Excitation of some of the posterior roots of the lumbar region of the spinal cord in dogs produces vesicular contraction. The excitation was caused either by means of an induced electric current or by mechanical irritation. 2. The vesicular contraction lasts longer than does the stimulus. The degree of the contraction depends on the condition of the animal and the stimulated root. 3. Every vesicular contraction is characterized by a latent period that seems to vary even with the same excitation and with other conditions (*Annali di Freniatria*, March 1904).

A CASE OF TUMOR IN THE FRONTAL LOBE (CEREBRAL GLIOMA).—M. L. LEBAR: The disease was not characterized by any marked disturbances. Seven weeks before death took place the patient complained of suffering from headache. Later there was mental obnubilation and uncleanness. During life the point of clinical interest was a decided exaggeration of the patellar reflexes and the presence of Kernig's sign on the side opposite to that on which the tumor was. The tumor was situated between the anterior part of the frontal lobe, the knee and the cauda of the corpus callosum and the base of the right frontal lobe (*Arch. Général de Médecine*, May 24, 1904).

SYPHILIS IN ART.—LAIGNEL-LAVASTINE: Art has long since seized certain pathological and hereditary traits and fixed it on canvas. Years later science verified the truth of the representations in art. Reference is made to some paintings by Goya, especially to No. 2571, in the Prado Museum. In this painting, the author claims, there is a realistic representation of the *nez en lorgnette* described as a characteristic shape of the syphilitic nose. Other stigmata of degeneracy are also masterfully represented and correspond accurately to those described by the scientist of to-day (*Nouvelle Iconographie de la Salpêtrière*, January-February, 1904).

GANGRENOUS FORM OF BROMISM.—DRS. HAL-LOPEAU AND VIEILLARD have reported this case to the Society of Dermatology and Syphilography of Paris. The patient, a woman, had been treated with bromide of potash during a period of some few days. The skin was covered with tape-shaped wounds arranged in more or less regular form. There were also some blisters and disseminated ecchymotic spots. This seems to have been an abnormal case of bromism (*Arch. Général de Médecine*, May 24, 1904).

RESEARCHES IN THE ANATOMICAL PATHOLOGY OF PARKINSON'S DISEASE.—DR. F. BURZIO: The main histological alterations found by the author in the cortical and spinal cells was chromatolysis. He also found atrophy and primary degeneration of the spinal tracts. The changes found strongly point to the presence of impaired alteration of nutrition and auto-intoxication in this disease (*Annali di Freniatria*, March, 1904)

SUTURING OF THE RADIAL NERVE.—DR. REYNIER reports several cases of suture of the radial nerve with subsequent recovery. Dr. Boekel obtained good results and restoration of function after the lapse of one month following the operation. In another case motor function was restored fourteen months after suturing the nerve. In Dr. Morestin's case the function was restored six months after the operation (*Progrès Médical*, May 14, 1904).

THE PATHOLOGY OF CHRONIC ALCOHOLISM.—DR. W. FORD ROBERTSON: The effects of alcoholic intemperance on the English people are much more grave and far-reaching than has generally been suspected. Chronic alcoholism has caused a diminished adaptability to circumstances. The spread of alcoholic poisoning of the nation should be checked by all available means (*British Journal of Inebriety*, April, 1904).

ABSORPTION OF SALINE SOLUTIONS IN THE INTESTINES.—DRS. CARNOT AND AMET: An 18 per thousand saline solution is rapidly absorbed by the intestines. When the saline solution is stronger it causes dilution by provoking excretion from the mucous membrane. This diluted saline solution is then absorbed. The weaker the solution injected the more readily is it absorbed (*Progrès Médical*, May 21, 1904).

EXPERIMENTAL EDEMA.—DRS. ACHARD AND PAISSEAU have obtained experimental edema by making intravenous injections forty-eight hours after ligation of the ureters. Subperitoneal edema is the first to appear, then follows intramuscular and finally cutaneous edema. Ordinary, saline or sugared water injected as explained produce edema (*Progrès Médical*, May 21, 1904).

EMOTIONAL FEVER.—DRS. TOULOUSE AND CL. VURPAS have observed three cases of emotional fever, the patients having presented temperatures of 39.8, 39.2 and 39 degrees C. respectively. Tachycardia is frequent in such cases, but disappears rapidly. The psychic consequences depend on the cases (*Progrès Médical*, May 21, 1904).

APPROPRIATION FOR THE NEW YORK STATE HOSPITALS FOR THE INSANE.—On May 9th, Gov. Odell signed a bill, by which an appropriation of \$3,203,000 was made for the use in the New York State Hospitals for the Insane.

THE SLEEPING DISEASE.—DR. DUPONT has had occasion to observe cases of the sleeping disease in three white persons. The disease is not confined to the negro exclusively (*Progrès Médical*, May 14, 1904).

BOOK REVIEWS.

LE BONHEUR ET L'INTELLIGENCE.—OSSIP-LOURIE. Félix Alcan, Paris, 1904. This work deals with abstract ideas regarding human happiness, pleasure and similar subjects in connection with our intelligence. According to the author, happiness necessarily exists because it is the prime principle of our existence. Pleasure is ephemeral, momentary, while happiness, according to human conception, should be something more durable. The conception of happiness, however, varies according to the individual, his surroundings, social status and mentality in general as well as in particular. Happiness derived from riches and worldly power are touched upon. While some of the author's views on this subtle subject are quite acceptable, he seems to lack broadness of view on matters outside of academic instruction on life in general. Speaking of the sentiment commonly called love, he criticises Janet for saying that a man in normal condition, physically and otherwise, reacts towards this sentiment according to his own free will. The author seems to be scandalized by this opinion. There are some interesting ideas in the chapters dealing with human power, slavery and the commission of suicide. He justifies the commission of suicide when existence becomes an unbearable burden, as by reason of sheer hunger, for instance. The suicide of Dumont, who accomplished so much for society by his philosophical works, one of which is entitled *Dépopulation et civilisation*, received nothing in return from society. He lived and worked as long as he had any belongings to exchange for food and committed suicide in all serenity because he could no longer exist,—all his belongings having been pawned or sold. The death of such a man and of many others like him under similar conditions is a slap in society's face; the social structure of to-day is such that all our energies are devoted to acquiring money, our tendencies to think and to attain a higher level being continuously strangled. The noble elements of society are the thinking men—the brain, so to speak, of the social organism. If society makes it impossible for this brain to exist, then this brain must perish and society must naturally perish in consequence.

The author agrees with Tolstoi that the feeling of fear of death is the result of a false conception of life. The continuance of life in a condition of inability to live intellectually is useless. Matter that lives an indefinite number of years is of low organization and death is a privilege characterizing organic superiority. Socrates did not fear death and Michael Angelo awaited death with all the serenity of an artist and poet.

A man does not ask to come into the world. Through blind circumstances he is placed in it. When his reason tells him that intellectual life is impossible for him, he has every right to take his own life.

False conception of happiness is the cause of unhappiness. Happiness consists not in having but in aspiring. The great mass of people does not know how to aspire and is unhappy. The few, the geniuses, who have the proper point of view of life, can enjoy it more in one day than the wealthiest man can in a life time. As an example of moments of beatitude of some great men, the author quotes Beethoven, who said: "I do not fear for the fate of my music. Its destiny cannot be negative; he who can hear it to the full extent will be delivered from the ills that others will keep dragging after themselves."

ESSAI SUR L'ANATOMIE PATHOLOGIQUE DES DEMENCES.—DR. HENRI-AUGUSTIN BRIDIER. Thèse Lyon, 1902.—From a study of the various forms of dementia, the author concludes as follows:

1. Dementia is always accompanied by some material cortical lesion.
2. From the anatomopathological point of view, no distinction can be made between the lesions found in organic dementia and those in purely psychic dementia.
3. Lesions found in dementia are characterized by marked alterations and disappearance of a large number of cortical cells.
4. The cellular lesions found in senile and in paralytic dementia respectively, are characteristic of themselves. The lesions of secondary dementias are recognized by exclusion and they differ one from the other in degree of cellular destruction.
5. Lesions of the cerebellum are frequently found in demented.

BOOKS AND PAMPHLETS RECEIVED.

A. Chapiet. Ectopie rénale.

Transaction of the Royal Academy of Medicine, Ireland, Vol. XXI., 1903.

Annual report of the Surgeon General of the Public Health and Marine Hospital Service of the United States for the fiscal year, 1902, Washington, D. C.

Transactions of the College of Physicians of Philadelphia, Vol. XXV., 3d series, 1903.

The Johns Hopkins Hospital Reports, Vol. XI., Nos. 1-9.

Dr. M. Lefèvre. Contribution à l'étude de la pathogénie des nevroses.

Drs. Antonin Poucet and M. Maillard. Rhumatisme Tuberculeux. (Pseudo-Rhumatisme d'Origine Bacillaire) Monographies Cliniques No. 34, 1903, G. Masson, Paris.

Dr. Donato Costanzo Eula. Proposte e Nuovil Studi in Antropologia Criminale. Cenno Critico ai Lavori del Congresso d'Antropologia Criminale di Amsterdam, 1903.

Prof. Sergio Sergi. Contributo allo Studio Anatomico-Clinico del Lemnisco Principale.

Dr. Percy Friedenbergl. Vicarious Menstruation into the Retina Followed by Detachment and Retinitis Striata. With Illustrations by the Author.

Report of the Superintendent of Indian Schools, Government Printing Office, Washington, 1903.

Dr. Hubert Richardson. The Treatment of Insomnia.

Dr. José Ingegnieros. Hipnotismo y Suggestion, 1903.

Drs. Eliseo Canton y José Ingegnieros. Locura del Embarazo.

Dr. Renold W. Wilcox. The Present Status of Iron Therapy. Nineteenth Annual Report of the Adirondack Cottage Sanitarium. Saranac Lake, 1903.

N. Vaschide and P. Rousseau. Contribution Experimentale à la Psychologie de la Perception de l'Espace.

Dr. B. S. Greidenberg. Subcutaneous Injections of Saline Solutions in Mental Diseases. Report, IXth Medical Congress, Russia, 1904.

Prof. Stéphane Leduc. Production du Sommeil et de l'Anesthésie Générale et Locale par les Courants Electriques.

Prof. Stéphane Leduc. Traitement Electrique de l' Hemiplégie.

Prof. Stéphane Leduc. L'Electrisation Cérébrale.

Prof. Stéphane Leduc. Etude sur les Courants Intermittents de Basse Tension.

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Nos. 3 AND 4.

TWO CASES OF FAMILIAL HEREDO-SPINAL ATROPHY (FRIEDREICH'S TYPE) WITH ONE AUTOPSY, AND ONE CASE OF SO-CALLED ABORTIVE FORM OF FRIEDREICH'S DISEASE.

ANATOMOPATHOLOGICAL AND CLINICAL STUDY.

BY PROF. G. MINGAZZINI, *Royal University of Rome, Italy*,
and
DR. G. PERUSINI, *Rome, Italy*.

(*From the School of Neuropathology, Rome, Italy.*)

(*Continued*).

II.—HISTOLOGICAL EXAMINATION.

Anatomopathology of the nervous system should be considered from the standpoint of the topography and the nature of the lesion. In our case we were enabled to study the topography of the lesions of the various tufts of fibres, but it was impossible to make a fully complementary cytological study of the lesion itself. The difficulty lies in the technique. Thus, the chromate salts, used in the preparation of the specimens, greatly hinder the study of the finer alterations of the nervous tissues. The methods of coloring generally used, such as the Weigert-Pal, van Gieson, carmine and the double coloring with hemotoxylin and fuchsine, are not of sufficiently recent date to answer the purpose for the study we had in hand. We have made serial sections of the spinal cord, the medulla oblongata, the cerebellum, the cerebrum and the nerve roots.

SPINAL CORD.—Along the entire extent of the spinal cord the pial meninges, the blood vessels and the connective tissues seem to be abnormally thickened. The tissue penetrates into the white substance in large septums and divides it into distinct portions. This division is made noticeable by the vivid fuchsine coloring of the connective tissue. These bands of tissue take an intense red coloring, when treated by the van Gieson method, and seem to be connective tissue. At all events, they seem to originate from the internal surface of the pia mater. It is difficult to determine whether the pia is more thickened about the roots and the posterior columns than it is elsewhere. If there is any difference, it is certainly very slight in degree. In the lumbar region the pia is perhaps a trifle more thickened than elsewhere. The meningeal thickening seems to end at the cervical region. In the lumbar region is noticed a thick septum that seems to be abnormal and that corresponds in position to the paramedian septum of the cervical region. Without making any positive assertions, it seems that the vessels of this lumbar region are more thickened than elsewhere. Among the other striking lesions is the complete obliteration of the central canal in its entire length up to the first cervical root.

LUMBAR REGION.—The condition of the meninges and blood vessels of this region has been considered above.

POSTERIOR COLUMNS.—It may be asserted that the entire territory of the posterior columns is the seat of slight degeneration. The few fibres that do remain are also markedly affected. Nevertheless, it is possible to follow out a certain gradation of the whole of the lesion, beginning with simple rarification and ending with absolute destruction of the fibres. Goll's is the most affected and Burdach's the less affected column. In Goll's column, in the peripheral zone, especially in the so-called *bandelette périphérique*, the fibres are absolutely destroyed. At the very most, sections prepared by the Pal method show some few myeline drops on a uniformly turbid yellow ground of sclerotic tissue. Nearer the limits of the ventral apex the fibres are found in larger quantities. No part of the posterior columns, however, can be said to be intact and many of the remaining fibres of the ventral apex show marked degeneration in sections treated by van Gieson's method and examined under high magnifying power. As for Goll's columns, properly speaking, their destruction did not allow of any distinction between their median and lateral parts. As has been remarked, Burdach's column was less affected than was Goll's column. The maximum destruction in Burdach's column corre-

sponded to the posterior radicular zone. The entire zone touching the posterior horn, on the contrary, was in relatively good condition. The areas that remained in comparatively good condition were the following: the cornu-radicular zone, the external *bandelette* of Charcot and Pierret and the lower limiting zone of Westphal. The cornu-commissural zone was in better condition than was the posterior-radicular zone. Of the gravely affected zones it is difficult to say which of them was more affected than the other. It may only be said that the cornu-radicular zone is in better condition than the external *bandelette* of Charcot and Pierret. It may be added that although the difference in question is quite noticeable, it is difficult to draw a distinct line of demarcation between the affected territories.

THE ZONE OF LISSAUER is distinctly degenerated in its internal as well as external parts, the degeneration being quite uniform in both.

ANTERO-LATERAL COLUMN.—The periphery of the entire antero-lateral column is the seat of a diffuse marginal degeneration. Although the latter appears irregularly, it delimitates to a certain extent the outlines of the column. The degeneration seems to be in direct relation with the thickened pia mater as recorded above. The irregularity of the degeneration is further confirmed by the fact that it does not appear in all the sections of the lumbar region. A coarsely triangular degenerated zone shoots off ventral to the posterior horn. This triangular area gradually spreads into a more extended area of slight degeneration or of simple rarification, loses its triangular form and finally ends in an ill-defined form. Such a zone occupies the external half of the crossed pyramidal tract, but it extends somewhat ventrad and invades also the fundamental band of the lateral column. The maximum degeneration does not correspond to the periphery, but is rather inward of the same. While the fibres are considerably degenerated, they are not absolutely destroyed in any one point. Some of the fibres in the centres of these zones are considerably reduced, but the lesion is certainly less marked than it is in the peripheral *bandelette* and in the postero-radicular zone. The remainder of the antero-lateral column is absolutely intact.

THE POSTERIOR ROOTS.—The posterior roots are appreciably degenerated, both in the extra-medullary portion, in which some scarce fibres still remain, and the endo-medullary portion that appears impoverished along its course through the spongy and gelatinous substances.

THE ANTERIOR ROOTS.—The anterior roots are, perhaps, some

what slenderer than normal. In the extra-medullary portion they are somewhat swollen.

THE GRAY SUBSTANCE.—The endo-cornual network is slightly impaired in the anterior and markedly impaired in the posterior horns. The cells of the various groups of the anterior horns seem to be well preserved and there does not seem to be any diminution in their number. They are well colored with fuchsine and carmine and the nuclei and nucleoli can be distinguished. The pericellular retraction spaces are abnormally large, but seem to be an artifact due to the action of the reagents used. The cells of the posterior horns, on the contrary, are poorly stained and diminished in number. Although the lesion of these cells is most evident, it is not easy to determine its nature. Besides the whole posterior horn seems to be shrunken.

DORSAL REGION.—The topography of the alterations found in this region is about the same as that described above. On the whole, the complexus of the degeneration in the territorial sense is also similar to that already mentioned. The lesions seem, however, to be more diffuse here. If there is any difference of lesion, it is that of degree. The difference between the degeneration in Goll's and Burdach's columns is far more marked here than it is in the corresponding columns of the lumbar region. In the dorsal region Goll's column may be said to be totally degenerated, including its ventral apex, while in the lumbar region the latter is partially preserved. Burdach's column is perhaps more degenerated in its inner side than is the corresponding area in the lumbar region. The lesion of the posterior-radicular zone continues here, but the limiting zone of Westphal is comparatively intact. The area corresponding to the cornu-radicular zone and the external *bandelette* of Charcot and Pierret are better preserved than is the latter. The lesion of the antero-lateral column is less marked in the portion that seems to correspond to the crossed pyramidal tract. In this region, however, it is more diffuse. In this region not only is the direct cerebellar tract affected, but the dorsal part of Gower's column seems also to be affected. The direct cerebellar tract is, perhaps, less affected than is the crossed pyramidal tract.

Nor does Clarke's column present any traces of cells or of fibrous network. This absence is noticeable even at the level of the dorsal roots, where under normal conditions they are quite prominent about the posterior horns. The other lesions mentioned do not present any abrupt changes worthy of mention.

CERVICAL REGION.—Marked changes are found in the area cor-

responding to this region. At this level the paramedian septum distinctly divides Goll's from Burdach's column. Goll's column is absolutely devoid of fibres and presents sclerosis, while Burdach's column is in comparatively good condition. The so-called ventral zone is not altogether preserved. The cornu-radicular and the limiting zone of Westphal are well preserved. The degeneration remains noticeable, although not too marked, about the posterior radicular zone. This variableness of degeneration of the ventral zone is seen even in sections made at close distances. The fibres of the crossed pyramidal tract are slightly rarified. These lesions are not symmetrical on both sides. The area corresponding to the direct cerebellar tract appears slightly degenerated in its marginal portion. This marginal degeneration or rarification makes it quite difficult to judge how far the impairment of the direct cerebellar tract extends. The cells of the anterior horns are well preserved, although the endo-cornual network is quite reduced. The posterior roots and the zone of Lissauer are degenerated. The degeneration is less marked, however, than in the lumbar region. The anterior roots are intact. Higher up in the cervical region, however, we could find slight rarification of fibres in the zone corresponding to the triangle of Helweg.

SUMMARY.—The degeneration of Goll's column is at its maximum in the cervical region. In Burdach's column there is a partial degeneration that becomes attenuated higher up, at the level where portions of the cornu-commissural and cornu-radicular zones are respected and where the ventral zone is variably degenerated. Clarke's column is disintegrated and there is moderate degeneration of the direct cerebellar tract. The latter cannot be traced with certainty above the first dorsal root. There is degeneration of the crossed pyramidal tract that cannot be traced beyond the level just mentioned. Higher up this degeneration probably continues in the corresponding zone; here, however, it presents simple rarification. Degeneration of Lissauer's zone and of the posterior roots. The latter reaches its maximum at the lumbar region. Slight lesion of Gower's column. Thickening of the pia mater, with which the slight diffuse marginal degeneration of almost the entire antero-lateral column seems to be in relation. There is no constant symmetry of lesion in both sides of the spinal cord.

We have found the special structure designated by Déjerine as "tourbillons." The latter are particularly well seen when treated with the carmine stain. The structures are well seen in transverse sections, but it is difficult to study them in relation to the

blood vessels in longitudinal sections. It is needless to give a description of the structures here, as Déjerine's description of them remains typical (see Fig. 9). The "tourbillons" are found particularly in the posterior columns, and especially in those corresponding to Goll's columns—where there is the maximum destruction of fibres. We did not find the "tourbillons" in the antero-lateral columns, even in places where these were markedly degenerated. With the van Gieson method the increased number of nuclei in the "tourbillons" is not seen. It may be asserted that there is neuroglial hyperplasia in the whole medullary periphery. In this periphery may be seen numerous nuclei and groups of *swollen* nuclei. In the nuclei are seen chromatic granulations. There are also multi-nuclear giant cells, the protoplasm of which is discernible. There are also typical spider cells. It is impossible to say what process caused the obstruction of the central canal.

We found tufts of fibrils in the periphery of the spinal cord, especially in the lumbar region. These tufts are immediately contiguous with the pia mater and seem to irradiate from a central nucleus. As we did not use the Weigert stain, it is impossible to say whether the tufts were offshoots of the pia or of neuroglial nuclei. With van Gieson's method they take only partially the red stain, while the connective tissue is well stained red (see Fig. 8). These tufts may have some bearing on the slight diffuse marginal degeneration of the spinal cord.

At the level of the first cervical roots and the lower limit of the medulla oblongata there is a visible degeneration of the posterior columns. This degeneration is much more marked in Goll's than it is in Burdach's column. In Goll's columns there are only a few fibres preserved. The ventral part of this column is in much better condition. At this level the pyramids seem to be intact. There is a slight diffuse marginal rarification of the fibres of the lateral columns. This rarification is quite evident in the areas corresponding to the fasciculus triangularis of Helweg. The gelatinous substance of the head of the posterior horns is slightly rarified. The roots of the Fifth pair are intact both at the level of the superior cervical and the inferior bulbar regions. Whatever remains here of the zone of Lissauer is rarified, but not degenerated. In the remainder of the medulla oblongata are found the following conditions: Degeneration of the nucleus of Goll's column and the cuneate fasciculus. The network about the fibres is in better condition in the nucleus of Burdach's column than it is in that of Goll's column. The cells of Goll's column are slightly visible, but those at the base of the nucleus of the cuneate fasciculus are more visible. The obliteration of the central canal ceases at the

lower limit of the medulla oblongata. There is a slight impairment of the condition of the tractus spinocerebellaris dorsalis and ventralis (antero-lateral ascending tract). The external ventral arcuate fibres are intact. The rest of the medulla oblongata is normal except for a slight rarification of the fibrous network in the peri-ventricular gray substance. All the nuclei of the bulbar nerves are normal. It cannot be said that the fibres here are more slender than normal. The fasciculus respiratorius is normal.

We have made sections of the entire mesencephalon, but have not found anything noteworthy.

The cerebellum is absolutely intact.

The cerebral cortex seems to be normal in so far as the methods at hand allow of examination.

Case III.—B. X., eighteen years of age, single, peasant. The mother died fourteen years ago of pleurisy with effusion. The father is living and is subject to hemophthisis. He coughs and is in poor physical health. He was twice married. With his first wife he had, besides the patient, a girl who is now eleven years of age. This girl can walk, but has generalized tremors of the head and seems to make incoordinate movements with her limbs. These movements increase when she is fatigued. According to the information given by the patient, this girl has also had an attack of rheumatism. A third child, born during the first marriage, died during infancy of an unknown disease. Five children were born during the second marriage. Three of these children died during infancy of unknown diseases and one died of diphtheria. The fifth child is living, but is cachectic, and the patient says that two years ago this child had "generalized swelling of his body."

When a child, B. X. had measles, typhoid fever and pneumonia. She walked well up to four or five years ago. At that time she had some febrile disease, that she says was rheumatism, that lasted about one month. She suffered from severe pains in the limbs, but it does not appear that she has had any articular pains. The pains are described as having been most severe, so that the patient could not move in her bed. The sister of the patient, who is subject to continuous tremors of the head, has had an attack of "rheumatism" precisely similar to that from which our patient has suffered. During the summer of 1903, the patient suffered from pains in the limbs. She experienced slight relief from stretching of the limbs. Menstruation was generally irregular and ceased entirely in 1903, but re-appeared in an irregular form later on. She was brought to the hospital November 5, 1903. Our examination of November 24 gave the following results:

OBJECTIVE EXAMINATION.—There was nothing of note about the cranial nerves in general. Monocular and binocular movements of the eyeballs were normal. When the patient looked at an object for a long time at a stretch, however, there was bilateral nystagmus in a horizontal direction. The facial and hypoglossal nerves seemed to be normal, there being no impairment of mo-

tility nor any tremors of the muscles supplied by these nerves. There was slight tremor of the head.

The upper limbs did not present any particular movements. There was no atrophy, except for some wasting in the left adductor pollicis muscle. When the hands were in repose, extended on the bed, they were animated by slight movements of adduction and abduction. There was nothing of note in regard to the passive movements. Active movements were all possible and were accomplished well. When the hands were extended together with the entire upper limb, they presented the movements described above. At the completion of the act of extension the hands also showed a tendency to assume a claw-shaped form. The muscular force was well preserved in the hands and in the forearms.

THE TRUNK OF THE BODY.—The thorax was more developed on the right than on the left side. The spinal column presented scoliosis, with the major curve directed towards the left.

The lower limbs did not present any particular movements, except for the left foot that had a tendency to drag, while the large toe was in hyperextension. Passive movements were enacted without encountering any resistance. The active movements of the thigh, leg and foot were performed slowly, and some of them were accomplished incompletely only. Among the incomplete movements were, for instance, extreme abduction of the thigh, lateral movements and dorsal extension of the foot. The muscular force was quite well preserved in the various segments of the limbs. When extended in bed, the patient succeeded quite well in assuming a sitting posture. She could not hold herself on her feet without the assistance of an attendant. When left to herself in the standing posture, she tried to maintain her position by enlarging the plane of support, but fell nevertheless. When walking she watched her feet, but fell, unless supported by an attendant. Even when led by attendants, she crossed one foot over the other while walking. It was impossible, therefore, to determine whether Romberg's sign existed.

REFLEXES.—The pupils were equal, of medium size and of regular outline. They reacted well to light and accommodation.

The superficial reflexes of the body in general were normal on both sides. The plantar reflexes, however, were impaired on both sides, the left one being the more impaired.

In the upper limbs, the radial, triceps, biceps and ulnar reflexes were absent. The patellar reflexes were impaired on both sides and more affected on the right side.

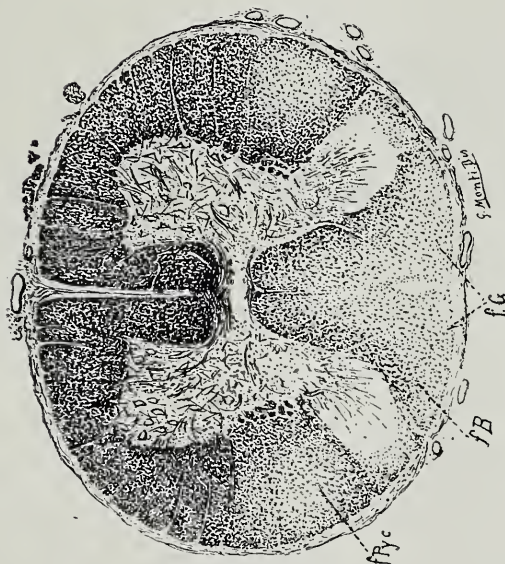


FIG. 2. Section at the level of the third lumbar root.
Abbreviations used for all the illustrations of the spinal cord :

- | | |
|---|--------------------------------|
| fG, Goll's column. | fcd, Direct cerebellar tract. |
| fB, Burdach's column. | fpyc, Crossed pyramidal tract. |
| cL, Clarke's column. | H, Triangle tract of Helweg. |
| A, Artery and anterior sulcus spinalis. | |

Seibert microscope. Ocular O, objective O.
Weigert-Pal method.

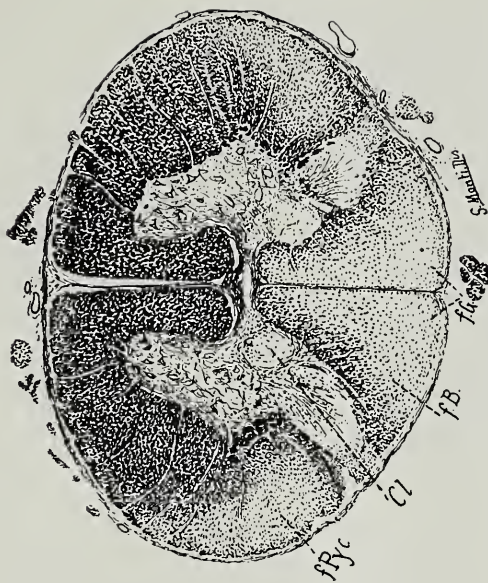


FIG. 3. Plane at the level of the seventh dorsal root.

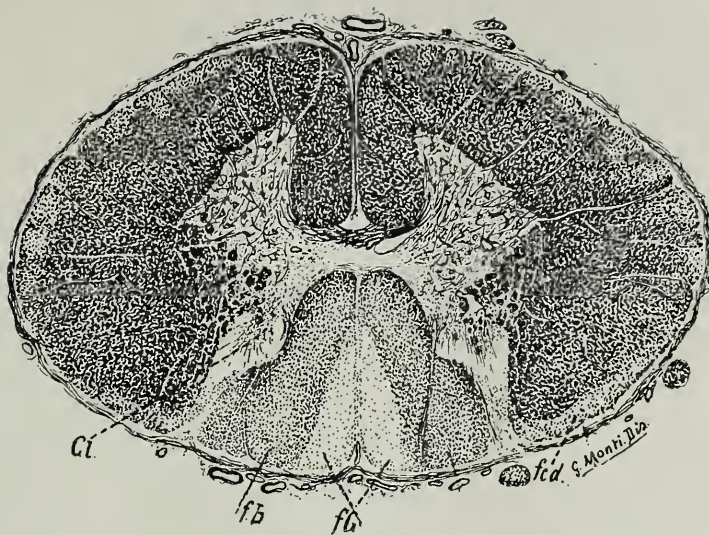


FIG. 4. Region between the first dorsal and eighth cervical roots.

Microscope and method as indicated in Fig. 2.

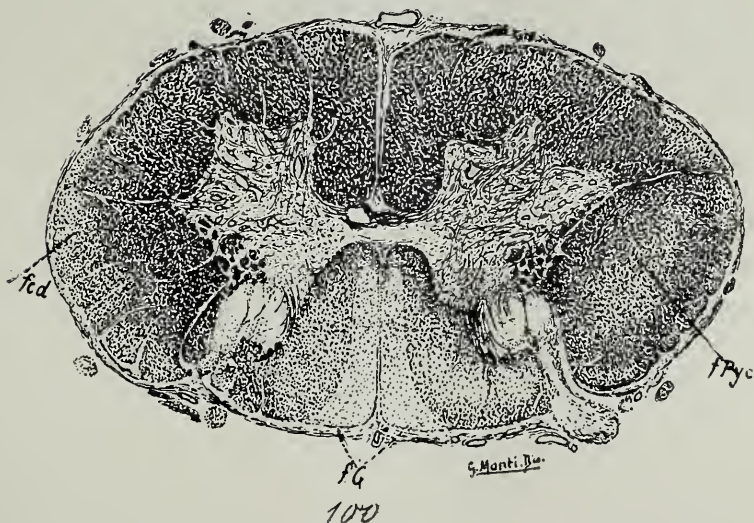
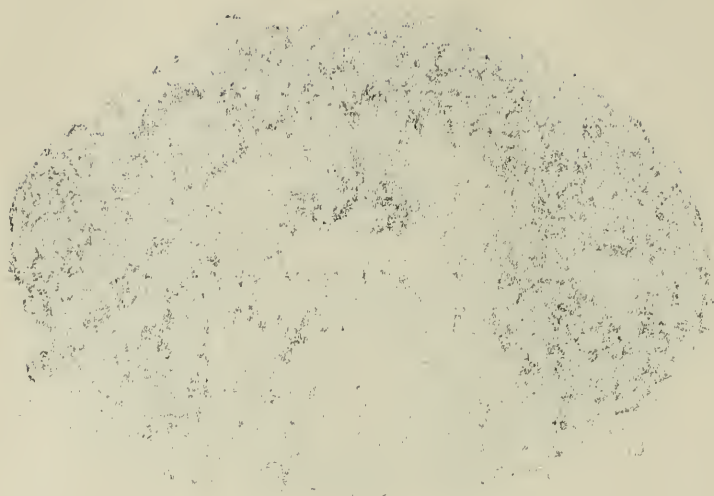


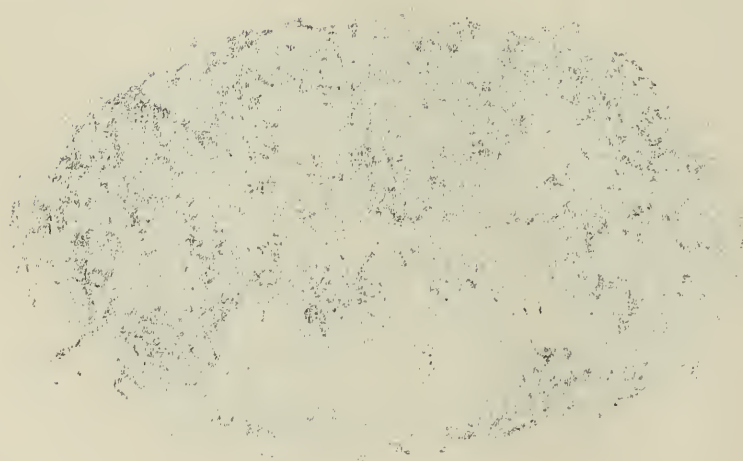
FIG. 5. Region between the seventh and eighth cervical roots.

Microscope and method as indicated in Fig. 2.



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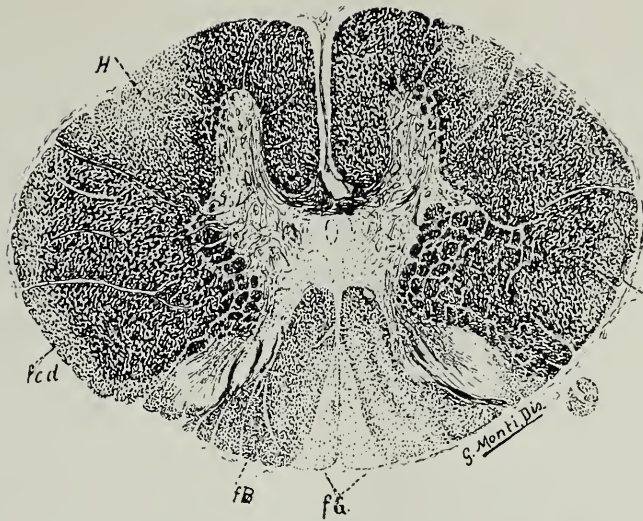


FIG. 6. Region at the level of the first cervical root.
Microscope and method as indicated in Fig. 2.

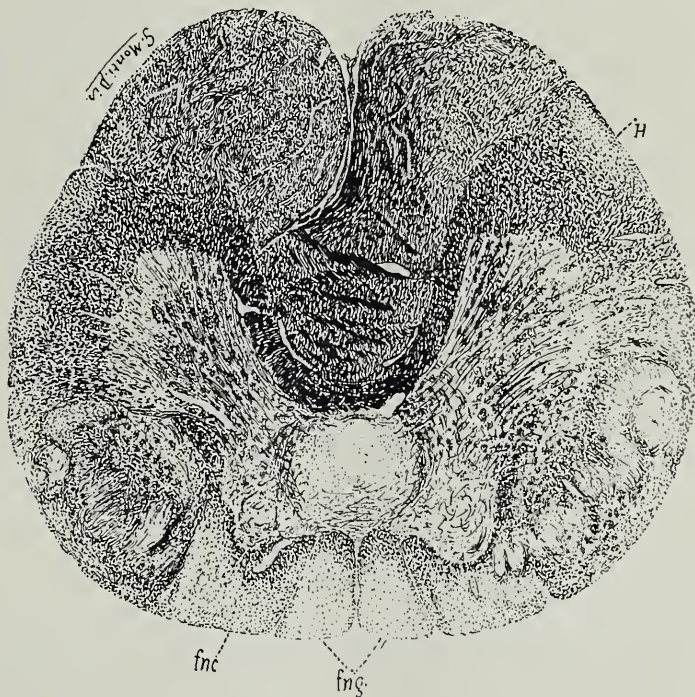


FIG. 7. Lower level of the medulla oblongata.
fnc, nucleus of the cuneate funiculus.
fng, nucleus of the funiculus gracilis

Microscope as in Fig. 2. Weigert-Pal stain
followed by fuchsine stain

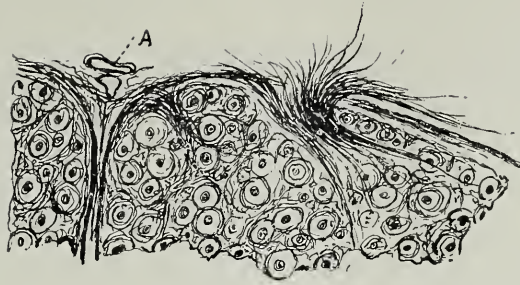


Fig. 8. Tuft of fibrils, in part probably of neuroglia nature, immediately underlying the pia mater in the region of the anterior median fissure.

Van Gieson's stain. Seibert microscope, ocular O, immersion lens $\frac{1}{12}$.

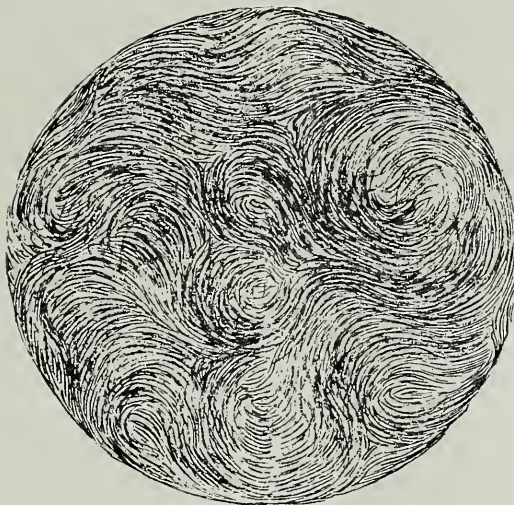


FIG. 9. "Tourbillons" in a section of the lumbar region corresponding to the median part of Goll's column.

Carmine stain. Seibert microscope. Ocular 1, objective 5.

SENSIBILITY.—Compression of the nervous trunks did not cause any pain. Tactile, thermic and dolorific sensibility was normal. The left side, however, was less sensitive to heat. The specific senses were normal. Speech was normal and there was no scanning of words.

MENTAL STATUS.—The physiognomy was expressionless. On the whole, it was not easy to judge from her looks that she was suffering from a severe disease. She seemed to be indifferent to her surroundings. Spontaneous and forced attention were below par. Mental perception was limited, and she showed no spontaneous manifestation of interest. She answered questions curtly and in a monotonous manner. Mental orientation as to time and space was sufficiently good. Her whole mental calibre was rather limited. It was difficult, therefore, to make out how much of the mental debility took place after the onset of the disease, and how much of it was the consequence of the affection.

III.—CLINICAL CONSIDERATIONS

Clinically, the French and other physicians accept Soca's synopsis and Ladame's definition of Friereich's disease. Soca's synopsis is as follows:

1. Ataxic gait (symptom of first importance).
2. Involuntary movements (symptom of importance).
3. Special deformity of the feet (symptom of prime importance).
4. Scoliosis (important symptom).
5. Nystagmus with normal vision, absence of ocular paralysis and intact iris reactions.
6. Impairment of articulate speech (symptom of prime importance).
7. Abolition of the tendon reflexes.
8. Absence of pain of any kind, and especially of flashing pains.
9. Integrity of the bladder and the urinary function.
10. Onset of the disease during infancy and the familial characteristics of the affection.

According to Ladame, the disease is characterized by a slow and progressive incoordination of the four limbs dating from infancy. The disturbance begins in the lower limbs, extends to the trunk and upper limbs, and finally affects the tongue, larynx and the eyes without causing any disturbances of general sensibility or oculo-pupillary anomalies and without being accompanied by flashing pains. The disease generally affects several members of the same family.

Schultze's synopsis does not differ much from Soca's. Although it has the advantage of presenting the symptomatology in a chronological order, the chronological order is not verified clinically.

If the autonomy of Friedreich's disease is accepted, it is not quite so easy to draw its limiting lines. Indeed, this problem seems to be entirely too vast to permit of any definite statements, and we shall limit ourselves to making a few general remarks. The definition and synopsis of the disease given above have only a schematic meaning. Thus, for instance, the characteristics mentioned in any one of the distinguishing traits in the synopsis may be absent by exception. The contradiction one faces here is obvious. It seems to us that the conception of Friedreich's disease as it stands to-day is a resultant of the rigidly framed conceptions that have preceded up to the present day. The conception of this disease has gone through an evolution similar to that which tabes has gone through. And so, the conception of to-day of Friedreich's disease stands in relation to the primitive limited conception of it as that of tabes of to-day stands in relation to the conception of ataxia of Duchenne. Speaking of the various forms of Friedreich's disease, Raymond said recently that there were three forms of this affection: an integral, abortive and hybrid form. He added that the study of the integral form should be completed by that of the abortive forms. Marie is of the opinion that the symptomatology of Friedreich's disease differs with every patient. This statement is somewhat similar to that made by Charcot. According to him, the form of a familial disease differs with the subject affected by it. The study of the hybrid form of the disease is of importance in so far as it relates to cerebellar heredo-ataxia. The study of the integral form of the disease we touch upon in our two cases, Maria and Robert X. The abortive form we shall consider in connection with our case, B. X.

We shall commence by the study of the relation of Friedreich's disease to the syndrome of Marie. In this wise we shall also present a clear illustration of the present conception of the disease in question.

RELATION OF CEREBELLAR ATAXIA OF MARIE TO FRIEDREICH'S DISEASE.

The syndrome known to-day by his name was described by Marie in 1893. He based his observations on sixteen cases of familial disease characterized by ataxic gait and cerebellar incoordination that he considered as being due to cerebellar lesions. Two

of these cases were those of Fraser, three of Nonne, eight of Sanger Brown and three of Klippel and Durante. Later on, Brissaud and Londe published a case of this disease, and finally Londe published an inaugural thesis treating of twenty-five cases of the affection. In this thesis are collected two of Erb's cases, two by Seeligmueller and four other cases by Sanger Brown. A bird's-eye view of these studies warrants the statement that Marie is prudent in his deductions. Firstly, he attaches absolute importance to the visual disturbances, and does not consider it correct to speak of Seeligmueller's cases, in which these disturbances are absent, on a line with the syndrome that bears his name. Secondly, he considers that the syndrome that bears his name and Friedreich's disease are possibly one and the same affection figuring under different forms, but are due to an identical initial heredo-degeneracy of the nervous system. In Friedreich's disease, he adds, a different system of nervous tracts may be affected, causing a difference between this disease and the syndrome known as cerebellar heredo-ataxia. The following year, Ribel, in a paper appearing after Marie's publication, treating of the differential diagnosis of Friedreich's disease, said that it was too early as yet to accept the new classification of the disease, and he did not commit himself regarding the cases reported by Fraser, Menzel, Nonne, Klippel and Durante and Sanger Brown. Londe was more explicit on the subject. Thus, Brissaud and Londe do not consider it necessary to strictly adhere to the differential data given by Marie. Brissaud and Londe, on the contrary, speak of a cerebellar heredo-ataxia not characterized by the classic disturbances of vision (dyschromatopsia, restriction of the field of vision, decreased visual acuteness depending on papillary atrophy)—a variety that would be characteristic of cerebellar ataxia only. This consideration creates a confusion in the points of differential diagnosis. And, indeed, it seems to us that Londe's differential diagnosis of the two forms of the disease seems rather incomprehensible. Thus, he claims that it is quite easy to make a differential diagnosis between Friedreich's disease and heredo-cerebellar ataxia. The differentiation is based mostly on the presence of spasmodic phenomena: exaggeration of the patellar reflexes and the presence of clonus exclude the diagnosis of Friedreich's disease. He further says: "Conservation of these reflexes probably excludes the same diagnosis; the great difficulty that faces one, however, is when a subject suffering from cerebellar ataxia of familial nature presents decreased reflexes. Such was the case of Senator that Schultze interprets with the author as being one of heredo-ataxia."

Looking back at this case, we notice that the author admits himself that Marie's type of the disease may evolve into Friedreich's disease. Also that in the initial stage of Friedreich's disease the knee reflexes may not be abolished and, finally, that the time of onset may be tardy in both forms of the affection. It is hardly necessary to repeat that the author admits the possibility of the absence of ocular disturbances in Marie's type. Yet he himself speaks of cases of Friedreich's disease with paralyses of the ocular muscles. Nor does the author admit that there are absolute differential diagnostic points between the two types of the disease as regards the trophic changes (scoliosis and deformity of the feet). From a clinical point of view, he considers both forms as being due to impaired equilibrium, and thinks it right to unify them in this sense. This reasoning, however, does not seem to be in accord with the preceding statements. Thus, according to Londe, in some cases the question is reduced to "generalized cerebellar ataxia and the differential point rests with the degree of the patellar reflexes." Elsewhere, however, he says that it would be more logical to consider Friedreich's and Marie's types respectively as two forms of hereditary ataxia in the largest sense of the word.

The whole matter is reduced to a play of words. And although Londe does not seem to have specified particularly the differential characteristics, he is privileged to consider Friedreich and Marie's disease as belonging to the same group. He must admit, however, that some of the cases remain unclassified. To put the matter in other words, it seems to us that whoever has tried to draw a distinguishing trait between the two forms of the disease has come across a great difficulty. A hasty examination of the positive and negative symptoms is quite convincing to this effect. Indeed, none of the symptoms are absolute. It is questionable whether the grouped symptoms are more characteristic of one type of the disease or the other—in the scholastic sense of the word.

To be more precise, we shall speak of the various types in particular. No special characteristic is given of the heredity or of the familial nature of the disease. In 1895, Brissaud claimed that the onset of Marie's type of the disease was much more tardy than was that of Friedreich's disease. This notion gives way in face of cases of Friedreich's disease with a tardy onset. Such cases are sufficiently numerous, and Bonnus has collected thirteen of them. The cases that suffice to confirm the possibility of occurrence of visual impairment and paresis and paralysis of the ocular muscle in Friedreich's case are: one of Gowers, in which there

was diplopia; one of Erlenmeyer and one of Mendel that presented convergent strabismus, one of Ormerod, with ptosis, one of Joffroy, with ptosis and diplopia, one of Rouffinet, with slight paralysis of the rectus internus, one recent case of A. Morselli, with ptosis of the left lid, one of Pritzche, with strabismus, one of Rouffinet, with narrowing of the visual field and papillary atrophy of slow development, Cohn's case, with atrophy of the optic nerves, Joffroy's case, with narrowing of the visual field, Lepine's case, with a sensation of a mist before the eyes, and Raymond's case, with left ptosis, paresis of the left rectus externus and narrowing of the visual field. Inasmuch, however, as, besides the classic type of heredo-cerebellar ataxia, created by Marie, there exists another type, without visual disturbances and paralysis of the ocular muscles, the differential diagnosis should be based on the condition of the patellar reflexes. If the diagnosis is difficult in the first instance, it is certainly much more so in the second case. One should be most cautious in drawing conclusions on the ground of the existence or non-existence of the patellar reflexes. Absolute exaggeration of the reflexes amounting to clonus would speak in favor of the spasmodic form, Marie's type, and absence of the same would indicate Friedreich's type. In reality, however, intermediary forms are not wanting. Thus, if Nonne's case is classed as one of heredo-cerebellar ataxia, it should also be borne in mind that it did not present any exaggeration, but simple conservation of the patellar reflexes. In Menzel's case the patellar reflexes were at first exaggerated and lost this character one month before death. In Klippel and Durante's second case and in Pearce and Swan's case the reflex was at first preserved, but it disappeared eventually. On the other hand, can it be said that exaggeration or conservation of the patellar reflex can in itself invalidate the diagnosis of Friedreich's disease? Clarke's case may present some disputable points, but this cannot be said of Mirto's case. Yet in both cases the patellar reflexes were preserved. These reflexes were also preserved in the cases reported by Gladstone, Hodge, Lop, Seiffer and Combes. Raymond presented to his students a typical case with preserved reflexes. Hence, when a case is reduced clinically to a cerebellar ataxic gait, as Londe would have it, the diagnosis would have to be based on the condition of the patellar reflexes. Such clinical diagnoses are certainly impossible. It remains for us to say a few words regarding the interpretation of facts under consideration. We reserve these remarks, however, for the chapter on physio-pathology. When treating of this subject we shall see that Marie's original conception of the disease has been modified to a given extent. Some of

the authors who have participated in this modification are Thomas and Roux and Déjerine and Thomas.

As regards our own opinions, we agree with those of Amouroux, Baumlín, Veraguth and others, that there are two forms of familial ataxia—Friedreich's and Marie's type. Between these two there are intermediary varieties, however. Raymond, who, in 1898, did not accept cerebellar ataxia as a clinical entity, considering it as a symptomatic manifestation of certain cerebellar diseases and who grouped cases with visual disturbances as hybrid forms of Friedreich's disease, has recently admitted the existence of intermediary types of the affection. Patrick opposes the notion of the autonomy of heredo-cerebellar ataxia. Magnus's, Paravicini's and Legrain's cases are difficult to classify. Lenoble and Aubineau consider their cases as special forms of heredo-ataxia, their particular characteristics being: 1, early onset of the ocular symptoms during infancy and their progressive evolution. 2, comparatively late appearance of the other symptoms. 3, tendency to assume some of the characteristics of Friedreich's ataxia. According to some authors, the characteristic symptoms in such cases speaking in favor of heredo-cerebellar ataxia are: the condition of the eye ground, tremor of the tongue and hands, slight exaggeration of the patellar reflexes and hesitation in performing movements in the dark. The symptoms in favor of Friedreich's disease are: unimpaired intelligence, hemicrania, slight spells of vertigo and abolition of the patellar reflexes. The signs common to both types are: nystagmus, deviation of the spinal column, integrity of subjective and objective sensibility and unimpaired sense of taste, smell and hearing.

Clarke reports cases of two brothers with similar symptoms: cerebellar gait, tremors of the hands, spasms and exaggerated reflexes of the lower limbs, Babinski's sign, lateral nystagmus, incipient atrophy of the optic nerve, arching of the feet and scoliosis. One of the brothers presented foot-clonus and the younger one had ptosis. Trauma seems to have been the exciting cause in both cases.

Margulies's case was a woman, 36 years of age, who had been ill for some years and two of her sisters, the grandmother, a maternal aunt and, perhaps also, the father, had suffered from a similar disease. The patient had scoliosis, ataxia of the head, trunk of the body and the extremities—especially of the upper ones. Slight spasms of the arms and deformity of the feet and exaggerated deep reflexes. There was a certain degree of mental defect and impairment of speech.

It may be recalled that Seiffer disapproves of the dogmatic divi-

sion of the two forms of the disease. According to him, both forms belong to one and the same group and in a few cases only differing symptomatologically, the difference being due rather to cerebellar than to spinal lesion. It is never possible to know, however, whether the cerebellum or the spinal cord is affected exclusively.

To sum up what has been said.—If the differential diagnosis between Friedreich's and Marie's type of the disease is based on 1, the presence of ocular disturbances, 2, conservation or exaggeration of the patellar reflexes, and 3, on the tardy onset of the disease, great difficulty accompanies the diagnosis. Indeed, ocular disturbances are met with in Friedreich's type, the patellar reflexes may vary considerably and the onset may be tardy. Taking all this into consideration and noting the possible early onset in Marie's type, the outlined differential points lose their clinical significance. Broadly speaking, the largest sense in which Marie's type may be considered is from purely anatomo- and physio-pathological points of view. Although it is not totally illogical to attempt a differential diagnosis between the two forms of the disease, it is not practicable to maintain such a distinction at all cost. It seems much more reasonable to consider the two forms of the disease as belonging to the same group. The existence of intermediary forms has been demonstrated, and what is true of Marie's type of heredo-ataxia with ocular disturbances is so much the more applicable to Brissaud and Londe's attenuated form.

THE SO-CALLED INTEGRAL FORM OF FRIEDREICH'S DISEASE.

The extreme variability of the form of Friedreich's disease does not allow of any absolute lines of demarkation. We shall not attempt, therefore, to draw these lines, contenting ourselves with an affirmative demonstration of the correctness of our diagnosis of our cases Maria and Robert X. In our demonstration we shall make reference to Soca's synopsis. If the latter is subject to criticism theoretically, it remains useful practically. Soca's synopsis remains classic in its outlines and we shall complete it with the newer data on the subject. It seems to us handy to consider the synopsis in its reverse order.

1. *Onset During Infancy and Familial Nature of the Disease.*—

In our two cases this characteristic is demonstrated once more, and all the members of the family affected with the disease fell victims to it at the same age. In both our cases the disease set in when the subjects were about ten years of age. In other cases the familial character and heredity may be wanting. In our cases,

however, the neuro-psychopathic heredity is marked. To this is added consanguinity of the parents, bringing to light the convergent degenerative potentiality of the parents. This consanguinity is of great clinical importance. Thus, in Veraguth's cases there is no neuro-psychopathic heredity, but there is consanguinity of the parents and both families have subjects afflicted with the disease. According to some, heredity has a specific importance. According to Olenoff, for instance, eighty-two per cent. of the cases are hereditary. Broadly speaking, however, the heredity is generally one of transformation, in the direct sense of the word,—affecting preferably the male members of the family. This heredity is essentially of organic nature. It may be spinal or cerebral (on the paternal side) or alcoholic, diathetic or tubercular on the mother's side. Progressive general paralysis and tabes in the progenitors is of special importance in this heredity. According to Olenoff, it is rather rare to find the parents affected with Friedreich's disease, yet such cases have been reported by Rutimeyer, Biro Gowers, and Everet Smith. Botkine's case may be doubtful. Gowers' case is, indeed, typical: the patient, mother and grandmother all suffered from the disease. Heredity in some branches of "Friedreich" families is too well known to need any comment. Among the authors who have treated of this subject are Dreschfeld, Latil, Ormerod, Rook, Vizioli and Esposito. Syphilis does not seem to have any influence on the genesis of this disease. Marie considered it as a probable cause, Thalwitzer doubted its influence in his second case and Bayet has demonstrated that syphilis is a causative agent. Two of Gasnes' cases were the offspring of an ataxic and paralytic respectively. Cases in which heredity and familial characters are absent are those recorded by Bezold, Bonnus, Amouroux, Levkovitch, Philippe, Letulle and Vaquez, Namack, Berdez, Carslaw, Cousot, Combes, Schwarz, Seiffert (three cases), some cases by Raymond, etc.

Our patients did not present any infectious diseases to which could be traced the onset of their affection under consideration. According to Demoulin, when the familial character of the disease is absent, it is generally preceded by an infection. The influence of an infectious disease is illustrated by cases reported by Katz, Rutimeyer, Luntz, Huet, Ormerod, Vizioli, Soca, Variot, Teissier, Baumel, Amouroux, Dreschfeld, Gilles de la Tourette, Musso, Petit, Demoulin, Bonnus, Freyer, Simon, Stein, Schoenborn, Combemale and Ingelrans, Mannini, etc.

Our case, Maria X., sustained a trauma, and some authors would attach much importance to the fact. Bramwell, Pearce and Swan, Clarke, and others, for instance, lay much stress on similar inci-

dents in their cases. As for ourselves, we do not see fit to consider such an incident in this light, for where is there a child that has not sustained some trauma?

2. *Integrity of the Bladder and of the Urinary Function.*—Generally speaking, our patients did not present any disturbances of the genito-urinary tract. The case of Teissier, Gilles de la Tourrette and Bradbury, that presented urinary incontinence, Friedreich's second case that presented urinary retention and Musso's case that presented difficulty in micturition are rather exceptions to the rule. In some cases there was polyuria, albuminuria, etc. Recently, Raymond claimed that incontinence of urine was rather frequent and could be considered as one of the earliest symptoms of the disease. Menstruation is generally irregular during the course of this disease, and our patient, Maria X., had irregular menstruation. The sexual appetite was normal.

3. *Flashing Pains.*—The lancinating pains were more marked in Robert X. than in Maria X. According to some authors, as Déjerine and Thomas, flashing pains are rather exceptions than otherwise, but there are numerous cases reported in which flashing pains were present. As for Brissaud, he claims never to have seen a case without these pains. According to Raymond, disturbances of subjective sensibility are almost always present. According to Bonnus, these disturbances are more marked in cases with a tardy onset. This fact rather contradicts Olenoff's assertion that the absence of flashing pains excludes the diagnosis of the affection. At any rate, it seems erroneous to suppose that the rarity of these disturbances is due to integrity of the peripheral nerves. The statement needs further confirmation.

Now and then are also noticed hemicrania and ophthalmic hemicrania.

We shall now consider the disturbances of the objective sensibility. While Maria X. presented only a slight hypoesthesia of the feet that seemed to have been due to trophic changes (cicatricial tissue of the feet), Robert X., on the contrary, presented quite marked hypoalgesia and hypothermia of the feet and legs. These disturbances decreased towards the upper part of the thigh. According to Soca, the disturbances of the general sensibility are quite frequent. This assertion may be somewhat exaggerated. Nevertheless, analgesia, hyperesthesia, anesthesia, anathermia and delayed sensation are frequently met with. Baumlin's case is illustrative of these conditions. In Bauchaud's case (twins) there was anesthesia and flashing pains. In the cases of Pritzche and Seiffer there were disturbances of the general sensibility. In

Biro's case there was hypoaesthesia of the legs and feet. There are still other cases on record. Gilles de la Tourette, Blocq and Huet also claim that the disease may be associated with hysteria.

The muscular, stereognostic senses and the sense of position of the limbs were intact in our patients. Electric reaction was normal. This condition seems to be the rule. The cases of Bonnus, Luntz and Guizzetti had disturbances of the muscular sense, Oppenheim's case presented disturbances of the stereognostic sense and A. Morselli's case presented disturbances of thermic and muscular sensibility and of the sense of appreciation of space. Finally, Cestan and Sicard have found, while studying nine cases, the presence of visceral disturbances. Among the specified disturbances are testicular and tracheal analgesia. Of the whole number there were four men and five women. Eight of the entire number presented tracheal analgesia. Three of the four men presented testicular analgesia. The man whose testicular sensibility was intact also had normal tracheal sensibility. According to these authors, the disturbances of the visceral sensibility may be found at any period of the course of the disease. The analgesias that have been studied for the first time and that are similar to those found in tabes should not be considered as being of hystero-organic nature. As lesions of the posterior columns enter into the picture of Friedreich's disease, it should be expected to find some tabetic symptoms. The nature of these symptoms, however, would need careful anatomico-pathological examination.

In our case, Maria X., tracheal analgesia did not exist.

Electric excitability was decreased in some of Friedreich's cases. In one of Biro's cases with muscular atrophy there was partial reaction of degeneration. This reaction was obtained in small muscles of the hands, in the anterior tibial and in the calves of the legs. In one of Schoenborn's cases there was reaction of degeneration in the left radial muscle.

4. *Abolition of the Tendon Reflexes.*—The tendon reflexes were abolished in both of our cases. When treating of the differential diagnosis between Friedreich's and Marie's type of the disease we made some remarks regarding the significance of the absence of the patellar reflexes. We shall now speak of the presence of Babinski's sign in our case, Maria X., in whom the sign was more marked on the more affected side. The statistical reports in regard to this sign are rather scant. Cestan found it in six of his cases, Seiffer in one of his seven cases; Schoenborn and Putnam also make mention of it, and Oppenheim, Déjerine and Thomas speak of it as a frequent occurrence.

5. *Disturbances of Speech.*—This is one of the most constant disturbances and the most appreciable of the whole series of the syndrome. Marie has drawn a happy parallel between the disturbances of the gait and speech in this disease. These disturbances existed in both of our cases and made their appearance early in the course of the disease. The characteristics were: slowness, difficulty and scanning in pronunciation, accompanied by ataxia. These are not different, however, from disturbances of speech found in other nervous affections. Some authors claim that the scanning speech, properly speaking, is not met with in this disease, although Raymond claims that it may exist. It seems impossible, however, to distinguish between this scanning and that found in other diseases. Erb claims that the voice may be bitonal on account of laryngeal ataxia.

6. *Nystagmus with Unimpaired Vision. Absence of Ocular Paralysis and Normal Pupillary Reaction.*—We have made above some general and special remarks regarding this subject. We shall now add a few words regarding the nystagmus. Robert X. presented the sign to a slight degree, while Maria X. had, during repose, nystagmiform movements rather than nystagmus, properly speaking. The nystagmus produced during repose, as in Maria X., is not of as frequent occurrence as is that accompanying extreme lateral movements. The nystagmiform movements are most frequently horizontal, but may be vertical and even rotary. At times, the movements pass unobserved on a superficial examination. If the patient is invited to look at a given object for a sufficient length of time, the movement can be discerned. It may be lost sight of, however, if the patient looks at an object that moves rapidly. The nystagmus is usually bilateral and less frequently unilateral. It may set in at any period during the course of the disease and may be tardy in appearance. Once it makes its appearance, however, it does not undergo any changes. The variation of the pupillary diameter seems to depend on the variation of accommodation, but not on a synchronous spasmodic action of the iris. It is difficult to say how many movements are made in a given time. They are quite numerous and vary with the individual. It seems to us that it is not reasonable to ascribe to this sign any specific significance in its relation to Friedreich's disease. The same sign is met with in various other affections of the nervous system. Rouffinet has ascribed great importance to the nystagmiform movements in this disease. According to him, the oscillations in this affection are ample but less numerous than in *sclérose en plaques*

and in defective refraction. It should be remembered that in these affections the movements are also most variable. According to Mannini, the nystagmiform movements are not necessary accompaniments of the disease. According to Schultze and his pupil, Offergeld, the phenomenon may be produced even in well persons. In Hunter's case, of quite a typical description, nystagmus was wanting. The same was true of the cases of Lop, Luntz, Pacheco, Seiffer and Edleston. The case of the latter author was at an advanced period of the disease.

7. *Scoliosis*.—Scoliosis was absent in both of the cases under consideration. When scoliosis is present, the convexity is more frequently on the right than on the left side. It is generally confined to the dorsal region and quite frequently there is also compensatory curvature. A combination of scoliosis with lordosis is rather rare; lordosis alone is still more rare and kyphosis is exceptional. The authorities disagree as to the cause of this symptom and the majority agree that the causes are multiplex. In her monograph, Dr. Roussel presents satisfactory arguments on this subject. Among the causes of scoliosis she gives rickets, vicious attitudes of the body, diseases of the spinal cord, hysteria, trauma, difference in the lengths of the lower limbs, affections of the thorax, etc. In the disease under consideration the factors are certainly multiplex. There are paralyses, atrophy, hypotonia, etc. While scoliosis may be one of the first symptoms of the disease, its absence does not invalidate the diagnosis of Friedreich's disease.

8. *Special Deformity of the Feet*.—Robert X. did not present any special postures of the lower limbs. The muscular masses in Maria's case were scant and flaccid but did not present any hypotrophy in relation to each other. The knees presented the knock-knee attitude and the feet were wide apart, their instep touching the sheet when the patient was on her back. The deformity of the foot was characteristic: the dorsal curve was exaggerated as well as was the plantar arch, the large toe was in a condition of hyperextension, while its first phalanx was flexed over the second. The position of the other toes was similar on both sides. The enumerated deformities were far more apparent when the patient assumed a sitting posture. During the entire course of the disease, the deformity could be seen in various gradations. In Pribram's case there was extensor contraction of the four toes with ventral flexion of the large toe. This deformity of the feet is not pathognomonic of Friedreich's disease, as remark Cestan, Allard and Monod. Its pathogenesis is obscure. According to Déjerine and Thomas, the condition is due to hypertonia of the periarticular

muscles and sometimes to contractures. Hence, according to circumstances, the disturbance may be compared either to that met with in hemiplegia or to that due to muscular atrophy.

9. *Spontaneous Movements*.—The “Spontaneous movements” were hardly perceptible in Robert X., but quite marked in Maria X., the latter presenting at the same time the special disturbances noted by Soca: intermittent nystagmus of the face, slight oscillations of the head, such as are used in making signs of affirmation or negation, and involuntary flexion movements of the lower limbs when the patient was in a horizontal position. It is hardly necessary to remark that the complexus of the movements expressed by oscillations of the head, fine tremor of the nostrils, lips and eyelids—representing the facial nystagmus—give a characteristic expression to the face. These fibrillary movements were not present at all examinations. If they were absent on some days, they could be found on others. The spasmodic flexion contractions of the lower limbs that are often seen during a subjective examination may be mistaken for voluntary contractions. These contractions are best compared to reflexes caused by tickling of the feet. These movements are rapid and comprize flexion of the leg on the thigh and that of the thigh on the pelvis. After this rapid flexion the limb is not maintained in position, but gradually falls back like a dead mass.

10. *Staggering and Ataxic Gait*.—Before considering the active movements of the lower limbs we shall make a few preliminary remarks. The movements considered in the standing and in the horizontal posture respectively require individual consideration. In Robert X., for instance, all active movements were possible and complete. When his thigh was flexed on the pelvis, however, the leg trembled when extended. Aside from this, however, there was no limitation of motility in the absolute sense of the word. In Maria X., on the contrary, all active movements of the lower limbs were abolished, save for some limited flexion. When some movements were still possible and the patient was examined in the standing posture the act was begun with a visible fatigue well colored with the ataxic nature of the gait. The patient was then unable to pursue his walk, tried to enlarge his basis of support by spreading out his feet, changing the centre of gravity from one foot to the other. The tremor of the head then spread over the body, the complexus constituting the static ataxia. The patient then fell, unless supported by an assistant. The character of the gait, when it is possible, is, according to Charcot’s happy definition, ataxo-cerebellar. Properly speaking, however, one definition

is not always applicable to varieties of the gait as they are encountered during the entire course of the disease. Thus, at first there is impairment of gait, then there is staggering gait akin to a drunkard's gait. The gait is then characterized by oscillations or may present a mixture of oscillations and spinal ataxia, which, on the whole, represents ataxi-cerebellar symptoms. This may be said to be "Friedreich's" gait. Gilles de la Tourette has given us a valuable graphic illustration of the gait in question. Later on in the course of the disease the ataxia gives way to absolute and permanent immobility. The various gradations of the gait are well illustrated in our two patients, Robert X. and Maria X., as has already been related.

As regards Romberg's sign, it seems to us impossible to determine with certainty whether it exists in Friedreich's disease. Among those who claim that the sign does exist in Friedreich's disease are: Biro, Luntz, A. Morselli, Rennie, Stern, Wickel and Kopczynski. Déjerine and Thomas consider it as an inconstant sign and Raymond says that it is not always absent. Among those considering the sign in the positive light are: Friedreich and Schultze, Dumon, Letulle, Land, Vaquez, Blocq and Marinesco and Philippe and Oberthuer (the latter report two cases). The diagnoses of all these cases were verified by the anatomical findings. Besides, Soca found Romberg's sign in thirty-seven out of fifty-seven cases. We think that the divergence of opinion on this subject is due to the mode of interpreting existing facts. Romberg's sign can be looked for only in the early stages of the disease, when the patient is still able to hold himself on his feet. And even then it is difficult to judge which of the movements constitute Romberg's sign and which are due to the choreiform movements. Later on in the disease this research becomes impossible for obvious reasons.

Remarks on Soca's Synopsis.—Cousot complains of the paucity of anthropological data in the studies of Friedreich's disease. It seems to us that the infantile type of the patients does not correspond to any established pathological type, but that it is due to retarded development caused by multiplex factors, such as rachitis, premature immobility, etc. Some patients, indeed, look younger than they really are. Marie X., for instance, who was 23 years of age, did not look older than 12 or 13 years of age. In this patient we found a distinct anemic murmur. Letulle, Vaquez and Smith lay much stress on the rôle played by congenital mitral stenosis in these cases. In Karplus's case there was also mitral insufficiency. Other patients have had tubercular complica-

tions. In Oppenheim's case respiratory disturbances were due to ataxic tremors of the respiratory and especially of the abdominal muscles. Fuerstner's case presented dyspnea and six of Seiffer's cases presented respiratory disturbances. We do not intend to enter into any discussions regarding these items, but simply enumerate them in so far as they can possibly apply to the study of our cases. Some cases present themselves with rare complications, as were those with muscular atrophy—reported by Joffroy, Déjerine, Biro, Vincelet and Hunter. Bauemlin's case presented atrophy and pseudo-hypertrophy. The cases of Chauffard, Combes and Schoenborn presented athetoid movements. Lepine speaks of anteropulsions in one of his cases and, finally, there was deafness in Hodge's case. These rare symptoms did not exist in our cases. As regards the atrophies, it suffices to call to mind Déjerine and Sottas' hypertrophic interstitial neuritis that resembles very much Friedreich's syndrome.

Both our patients present disturbances of handwriting, consisting of tremors and imperfect tracings. In Maria X. this disturbance appeared quite early in the course of the disease. These disturbances are not, therefore, dependent on the ataxia of the upper limbs. According to Charcot, the hands of these patients drop upon the object they wish to take hold of as a bird of prey falls upon its victim. This particular sign was not evident in Maria X. Nor did our patient present the particular deformity of the hand on which Cestan and Sicard and perhaps also Friedenreich lay so much stress. In eight of their nine cases, Cestan and Sicard found the deformity of the hand that they describe as "main bote." This form is particularly apparent when the patient is attempting to take hold of an object while the upper limb is extended. According to the authors cited above, the pathogenesis of this deformity consists of a certain degree of local paresis of the interosseous and lumbrical muscles, without affecting the muscular movements of the forearm. There are neither tendon retractions nor impairment of the articular surfaces. There is a correspondence between the deformity of the hand and that of the foot and the deformity of both is in direct relation with the evolution of the disease.

Researches into the cerebro-spinal fluid in Friedreich's disease have thus far remained negative. Sicard has made three examinations. Barjon and Cade examined the cerebro-spinal fluid at an autopsy of a "Friedreich" case and found a reduction in the cellular elements. The liquid contained red blood corpuscles in particular, some lymphocytes but no leucocytes. The patient died of pneumonia, and pneumococci were found in the liquid in question. The meninges did not show any signs of inflammation. There

were signs of an old diffuse cerebral pachymeningitis that seemed to be the result of a common chronic inflammation. In our cases it was impossible to examine the cerebro-spinal fluid.

The mental status of our patients does not add any important information to our study. The mental depression that existed was in accord with the affliction to which Maria X. was subject. Robert X's mental status was quite limited. According to Soca, the imbecility of these patients is only apparent and the expressionless physiognomy is due to impaired muscular tonicity. The accompanying disturbance of speech gives an impression of imbecility. The cases of Nolan, Batten, Power, Vincelet and two cases of Pritzche presented symptoms of idiocy, imbecility and had delusions of persecution. Szczypiorski's case was an imbecile and epileptic. Two of Seiffer's cases presented slight psychic disturbances. According to Soca, these patients have a special psychic status. Gilles de la Tourette, Blocq and Huet compare their mental status to that found in patients with *sclérose en plaques*. These patients are said to have spasmodic laughing spells. Two of Seiffer's seven cases presented these laughing spells. Ballet considers Friedreich's disease as a cause of mental affections. These facts are of significance, lessening still more the attempted line of demarkation between Marie's and Friedreich's type of the disease. Indeed, it has been claimed by some that Marie's type was characterized by special mental disturbances. Seeligmuller went so far as to designate these disturbances as mental ataxia. Among other cases, that of Nonne and the 12th case of Sanger Brown presented grave mental disturbances and even imbecility.

Our case, Robert X., died in diabetic coma, like the case of Best. In other cases death took place suddenly. Guizzetti thinks that in such cases there are frequently myocardial and pneumo-gastric lesions.

Considering what we have said about our cases Robert X. and Maria X., we feel certain that both patients suffered from Friedreich's disease. The clinical characteristics of both cases were as follows:

1. Onset of the disease during infancy and familial character of the disease.
2. Integrity of the bladder and urinary function.
3. Abolition of the tendon reflexes.
4. Disturbances of speech.
5. Absence of ocular paralyses. Normal pupillary reaction and nystagmiform movements.
6. Spontaneous movements.
7. Staggering and ataxic gait and evolution of the morbid form.

In addition to the above enumerated characteristics Maria X. presented special deformity of the feet. This adds to the full clinical picture of the affection. On the other hand, the absence, both in Maria and Robert, of scoliosis, the presence of slight pains and still slighter disturbances of sensibility do not invalidate the diagnosis.

(To be concluded).

A CASE OF LEFT HEMIPLEGIA WITH RIGHT HEMIANESTHESIA OF TRAUMATIC ORI- GIN WITHOUT ORGANIC LESION OF THE SPINAL CORD.

BY DR. PIETRO TIMPANO.

The case I am about to describe presents a grouping of symptoms singularly similar to Brown-Sequard's syndrome, with this difference, however, that the syndrome is *inverted* in this case: the anesthesia was on the side on which the trauma took place while the paralysis was on the opposite side. Besides, the trauma that had caused these disturbances *did not cause any lesion* either of the spinal cord or of any important nerve trunks.

The history of the case is as follows:

M. A., twenty years of age, driver. His father was an alcoholic and died of pneumonia. The mother is healthy. The patient has two brothers and three sisters who are healthy. When a child the patient had measles. Outside of this he always enjoyed good health. He denies having had syphilis, does not use alcoholic drinks and smokes moderately. April 29, 1904, he engaged in a quarrel with a fellow-driver that ended in a fight, in which he received a wound inflicted with a knife. The wound was located in the right side of the neck. On receiving the wound the patient fell. He did not lose consciousness, but could not get up because the left side of his body was paralyzed. He was taken to the hospital in Rome.

The wound on the right side of the neck was about two fingers' breadth distant from the spinous apophyses of the last cervical vertebra. The wound was directed obliquely from above downward and from inward outward. It did not penetrate into the

spinal foramen. The whole right side of the body was paralyzed and there was anesthesia of the left side of body. The patient left the hospital when the wound was healed, but the paralysis and anesthesia remained as described above.

OBJECTIVE EXAMINATION.—The results of an examination made fifteen days later are related below. The pupils reacted normally to light and accommodation. The visual field was normal on both sides. There was nothing of note in regard to special sensibility. The patellar, abdominal, thoracic and pharyngeal reflexes were normal. The plantar and the deep reflexes of the upper limbs were impaired. Active and passive movements of the left upper and lower limbs were considerably limited. The muscular movements were normal on the right side. Thermic and dolorific sensibility was noticeably diminished on the right side of the body. The hypoesthesia reached anteriorly to the upper border of the third rib, laterally to the upper margin of the fourth rib and posteriorly two fingers' breadth above the spine of the scapula. Below this hypoesthesia involved the whole lower limb. Above the limit of the zone of hypoesthesia there was a zone of hyperesthesia. The latter zone was more marked posteriorly than anteriorly. There was no muscular atrophy and reaction of degeneration was absent.

The patient was subjected to treatment of various forms,—hygienic, mechanical and hydroelectric. He made a progressive and complete recovery. Many similar cases can be found recorded in medical literature.

Brunelli reported a case in which the patient received a wound near the last dorsal vertebra. The wound did not penetrate into the spinal foramen. This wound was immediately followed by paralysis of the right lower limb and anesthesia of the left lower limb. Among others, Gatti, Paladini and Sciamanna have also reported similar cases. Sciamanna has had occasion to study several cases in question, one of which is very much similar to my own, and I shall briefly cite it below.

“P. T., twenty-five years of age, shoemaker. He has never suffered from any diseases of consequence and his heredity is negative. One day he was passing through a street when suddenly a man sprang at him and cut him with a knife, inflicting a wound in the left supraspinous region of the scapula. He fell on his back as soon as the wound was inflicted. He immediately tried to get up, but found it impossible to do so. As he fell, a knife that he was holding in his hand dropped out of it beside

him. As he was lying helplessly on the ground he tried to grasp the knife that had fallen out of his hand, but the hand was now limp and he could not take hold of the knife with it. The patient was transported to his home. As soon as he was brought to the house it was found that he could not move the right lower limb or open and shut completely the right hand. The entire upper right limb was powerless and the patient had a tingling sensation in it. A few days later while looking at himself in a mirror he noticed that the right eye-lid was drooping.

“OBJECTIVE EXAMINATION.—The pupils were equal and reacted normally to light and accommodation. The visual field was normal on both sides. The facial nerves and the special senses were normal. The pharyngeal reflex was somewhat impaired. The conjunctival, thoracic and abdominal reflexes were normal. The patellar reflexes were exaggerated, while the plantar reflexes were normal. A cicatrix on the left side, above the spinous process of the scapula and parallel to the shoulder line indicated where the wound had been. The lower end of the cicatrix almost touched the line corresponding to the spinous process of the scapula.

“Motility of the left upper limb was normal. Extension of the right hand was impossible unless the fingers were flexed so that the hand assumed the shape of a closed fist. When this shape was assumed it became absolutely impossible to flex the hand over the forearm. The muscular force of the right hand was also diminished. When extended the whole right limb presented a slight tremor. The movements of the right lower limb were impaired and there was a slight degree of muscular atrophy. In the left limb motility was normal. Galvanic and faradic reaction was normal in all the limbs.

“SENSIBILITY.—There was a zone of hypoaesthesia on the left side. The limits of this zone were: from the median line anteriorly to the median line posteriorly; superiorly the limiting line was one that could be drawn through the nipple, the axilla and the ridge of the spinous process of the scapula; below the zone involved the entire extremity. In the arm the hypoaesthesia involved the inner side. Besides, there were areas of hypoaesthesia of the right upper limb. These areas were limited to the inner surface of the forearm, the back of the right hand and the entire surface of the little finger.”

What interpretation is one to give to this special nervous manifestation? Formerly it was considered as an essentially hysterical phenomenon. And even to-day a good many authors consider it as a proteiform neurosis.

From the cases cited above and from those cited by different authors it is seen that they all have a major characteristic trait: a lesion of the neck, superior and posterior part of the body,—more or less near the spinal column—without lesion of the spinal cord—is immediately followed by a syndrome analogous to the syndrome of Brown-Sequard. In Brown-Sequard's syndrome, however, there is always unilateral lesion of the spinal cord. Besides, in the syndrome I am about to consider the anesthesia is manifested on the side where the lesion was inflicted, the paralysis is on the opposite side and the spinal cord is in no wise implicated—so far as investigation can reveal. These facts would lead one to suppose that possibly the genesis of the neurosis that interests us is different from the other traumatic neuroses. Or, again, that in these special cases the pathogenesis should not be looked for in the altered psyche of the patient but rather in the functional disturbance of the spinal cord induced by the trauma of the given region.

Leyden and others tried to explain this syndrome by a supposition that a centripetal neuritis took place that gradually ended by altering the spinal cord. This assumption naturally excludes the possibility of a *functional lesion* and rather fixes the consideration on an *anatomo-pathological lesion* properly speaking.

Gravès thinks that the mechanism by which these paralyses are produced is similar to that which characterizes the production of intestinal paraplegias. The inflamed mucous membrane, he explains, stimulates the nervous fibres, that in their turn transmit the stimulus to the spinal cord; the latter finally reacts in a manner that results in disturbed motility of the lower limbs. Stanley gives a similar interpretation of such facts and as an example cites cases of paralyses of the lower limbs dependent on lesions of the urinary tract; the crural and sciatic plexus are acted upon by way of the lumbo-sacral plexus.

Sahmans tried to reproduce experimentally in animals symptoms of shock by striking the spinal column with a hammer. Although the experimental conditions and those found in man after a traumatism in question are not identical, the author found, nevertheless, that symptoms of shock could be obtained experimentally without there being any appreciable anatomical lesions either macroscopically or microscopically. In three cases of spinal concussion in man the author did not find any anatomical changes of the spinal cord. In one case the autopsy was performed six weeks after the accident, in one two months and in one eight months after the accident.

Thus it is seen that a lesion without involving the spinal cord

may impair more or less severely the function of some of its segments. This impairment is enacted through a series of abnormal stimuli proceeding from the point wounded to the neurones.

Can similar conditions explain the syndrome we are here considering? Certainly the supposition that similar conditions are possible is not unfounded. Some objections may be made against this supposition. One is that the syndrome is not verified in all cases in which a traumatic wound is inflicted on the neck or in other parts near the spinal column. Another objection is that the syndrome under discussion should not be considered as an entity, but should rather be looked on in the light of a hysterical manifestation: the great neurosis is so many-fold and complex that one should not wonder when it assumes the form of a symptom-complex so well defined and distributed as is the Brown-Sequard syndrome.

Replying to the first objection, I should say that if some individuals, on receiving a traumatic wound in the vicinity of the spinal cord, do manifest the syndrome under discussion and others do not,—it remains for us to conclude that the nervous system of those who do manifest the syndrome is especially predisposed to the morbid manifestation; a peripheral lesion gives rise to a special grouping of stimuli that reach the spinal cord and induce a functional modification that is characterized by more or less permanent abolition of motility and sensibility in given regions of the body.


I shall now consider the second objection. While one readily admits that hysteria is a proteiform neurosis, there are certain constant facts in relation to our syndrome that should be borne in mind. Thus, a peripheral lesion in the vicinity of the spinal column very frequently gives rise to a syndrome characterized by a morphologico-functional disposition (although inverted) similar to that observed by Brown-Sequard in cases of unilateral lesion of the spinal cord. Besides, this syndrome has never been observed to take place when the lesion is far from the spinal column. Adding to what precedes the absence of hysterical stigmata in my case and in that of Professor Sciamanna, the impossibility to cure such syndromes, at least to modify them to any degree by psychic means, leads one to conclude that the mechanism of the genesis of the syndrome is altogether different from that characterizing hystero-traumatisms.

What, then, are the conditions that bring about the syndrome I am considering?

In my case there is hemiplegia with hemianesthesia on the opposite side. Professor Sciamanna's case presented similar symp-

toms. In both cases the wound did not extend into the cavity of the spinal canal. If an organic lesion is excluded in these cases, as it should be, one is forced to admit that there must have been produced some interruption along the motor and sensory tracts in given segments of the spinal cord. It becomes necessary to accept the hypothesis, therefore, that a peripheral lesion may cause functional disturbances at a distance. The question arises, however, whether a stimulus started peripherally can interrupt the motor and sensory conduction of the spinal cord in one particular point as a hemi-section of the spinal cord at a given level would do? Evidently this cannot be. In order to understand the *modus operandi* of the functional disturbance at a distance it is necessary to suppose that the stimuli run along the ascending tracts and, on reaching the first group of cells in which they are in communication, discharge the stimuli into these cells; the latter, in their turn, transmit the stimuli to another group of cells in the neighborhood, etc. Under these conditions various medullary segments may become involved.

To understand these conditions more clearly I present here a schema of the tracts that the peripheral stimuli must cover in order to produce the syndrome under consideration. I may add that Professor Sciamanna admits without hesitation that this schema is a plausible one.

It is known that many posterior radicular fibres, after penetrating into the spinal cord, divide into ascending and descending branches. This division may be represented by a figure like this . Both the ascending and descending branches run vertically a certain distance then bend at an angle and penetrate into the gray matter where they divide into terminal branches and surround the cells. Both the ascending and descending branches give off collateral branches that penetrate into the gray substance and sub-divide in their turn into other branches. The other fibres penetrate immediately into the gray substance. Before penetrating into the gray substance, however, they also give off collateral ascending and descending branches that in their turn subdivide into terminal branches and finally surround the cellular elements of the gray substance.

An irritation that runs through one or more of the spinal roots may also be transmitted along the respective descending branches and thus cause functional impairment in some of the lower spinal segments that communicate one with the other by means of these very branches. Thus, all further transmission of the normal stimuli that usually run along the posterior radicular tracts to the brain may be interfered with. Under these conditions the func-

tion of the inferior spinal segments only, the posterior roots of which had been irritated, becomes impaired. These conditions give rise to hemianesthesia on the same side on which the lesion had been inflicted. The presence of the zone of hyperesthesia above that of anesthesia may be explained somewhat differently. Thus, it may be assumed that the same stimulus that has succeeded, either through the special quality of the fibres or some conditions unknown to us, in producing grave functional impairment in the descending branches of the posterior roots has also caused an irritation in a given area only of the ascending branches that have become hyperexcitable without having sustained a shock properly speaking.

The paralysis on the side opposite to that on which the lesion is may be explained in a manner given below.

Thus, the two halves of the segments of the spinal cord are connected by means of endogenous fibres coming both from the medullary and commissural cells around which terminate the ramifications of the collateral branches of the posterior radicular fibres; besides, the endogenous fibres themselves divide into ascending and descending branches with collateral branches of transverse direction that in their turn ramify around other commissural cells; these cells are, in their turn, in connection with the anterior radicular cells of the respective segments. Under these conditions it may be assumed that the same irritation that comes from the opposite side may affect the cellular elements of the various lower segments and impede the passage of a normal stimulus coming from the cortex and through the direct and crossed pyramidal tracts. In this manner paralysis on the side opposite to that on which the lesion is inflicted is easily explained.

From what precedes it would seem that the motor and sensory disturbances should be manifested in the various spinal segments, the highest of which should be on a plane inferior to that of the seat of the peripheral lesion.

Naturally this explanation is founded on a hypothesis only. I do not presume to present in this a positive explanation of the syndromes under consideration. I simply wished to direct the attention to the possibilities as explained above because many facts in connection with similar syndromes warrant the conclusion that the latter are of functional rather than of hysterical nature. While awaiting the results of ulterior studies and more precise methods that will shed more light on the subject here considered medico-legal science will have to rely on the meagre knowledge we possess at present.

As I have built hypotheses on the supposition of the action of

nervous shock, I shall now consider the *modus operandi* of functional disturbance of the spinal cord due to shock.

According to Brown-Sequard, abnormal stimuli reaching the spinal cord may cause vascular constriction of the pia mater and of the spinal cord itself. The resulting poor nutrition may cause paralyses. Karow also thinks that the *modus operandi* of shock may be explained by vaso-motor paralysis limited to a given area in the spinal cord. Admitting this condition, it is difficult to understand how the spinal cord can undergo a spell of anemia or hyperemia for a more or less marked period of time without suffering any anatomical alterations.

Jaccoud applies the theory of over-work of the cellular elements. A strong and prolonged stimulus may lead to cellular invalidity so that there can be no reaction to normal stimuli. Dupley and Obersteiner accept this theory.

Valentin has demonstrated that a nerve excited with a rather strong continuous electric current becomes incapable of conducting a stimulus applied above the electrified area, whereas normally such a stimulus gives muscular contraction. Mattenci, Pfeuger, Dubois-Reymond and others verified the results of this experiment.

Jaccoud wants to demonstrate that similar conditions take place in the spinal cord. On passing a strong galvanic current into the spinal cord the latter became incapable of responding to another electric stimulation. The author then assumed that the first electric current probably caused a modification of the molecular status and chemical constitution of the cellular elements of the spinal cord.

While we have no means of demonstrating the conditions named above, their presence may certainly be accepted as a fact when the cellular elements are subjected to abnormal stimuli as explained above. The studies of Van Gehuchten, Marinesco, Lugaro, Pergens and others facilitate the understanding of the question.

According to Pergens, the nucleine of the retinal cells becomes liquid during the initial stage of functional activity. During the stage of functional activity, properly speaking, the entire mass of nucleine becomes dissolved and finally consumed. Fatigue sets in when there is excessive consumption of the nucleine.

According to Mann and De Moore, the state of activity of the nervous cells in general is characterized by augmentation in volume of the protoplasm and decrease of the chromatic substance. Lugaro found that nervous cells react to peripheral lesions by disaggregation of their chromatic substance that may last a more

or less marked period of time and may also abolish the functional activity of the nervous cell itself.

It seems that the rôle of the chromatic substance is that of accumulating and developing energy by means of chemical changes that are set into action during the passage of a nervous wave. Hence it is readily conceivable that a normal stimulus should leave the nervous cellular activity intact while an abnormal stimulus should impair the anatomical and functional activity of the cell or even abolish its functional activity and thus make the transmission of normal motor and sensory waves impossible. It can readily be understood that such abnormal reaction varies with the individual subject.

According to Lugaro, Marinesco and others, the excitability of the nervous system in neuropathic subjects depends on various conditions, such as repeated emotions of sudden onset and marked degree, chronic intoxications, circulatory disturbances, etc. Under such conditions the nervous elements are predisposed to ready disaggregation of their chromatic substance that is needed for their nutritive and functional equilibrium.

For the reasons presented above it seems rational to suppose that the mechanism of the genesis of the syndromes considered in this paper does not depend on a functional psychic disturbance, but is rather due to a local disturbance of the spinal cord.*

* The illustrations for this paper reached here too late to be inserted.—Ed.

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THE FUNCTION OF THE THYROID AND PARATHYROID BODIES.

The function of the thyroid and parathyroid bodies is of great interest to-day. Many important papers have been published on this subject and the application of the knowledge thus furnished has been of important practical value in medicine. One of the most valuable papers on this subject that has come to our notice is that written by Professor Alfred Rouxeau. The paper is entitled "La thyroïdectomie et l'extirpation des glandes parathyroïdes chez le lapin" and was published in the *Gazette médicale de Nantes*.

While the results of Professor Rouxeau's experiments are not startling in novelty of discovery, they are important from the standpoint of accuracy. As is well known, experiments on the thyroid and parathyroid bodies are delicate of handling. The conclusions drawn from such experiments depend largely on the experimenter's conscientiousness. The striking feature of the

paper here considered is the conscientiousness of method, procedure and conclusion. This preciseness of investigation is of particular value to-day, when therapeutic application of the thyroid body is being made in many and varied ways.

A detailed account is given of the experiments made on rabbits. Thyroidectomy was practiced in forty-five cases. Thyroidectomy and simultaneous extirpation of the parathyroid glandules was performed in fifty-two cases. Thyroidectomy and consecutive ablation of the parathyroid bodies was performed in thirteen cases.

Some of the general conclusions arrived at are:

1. Thyroidectomy is not followed by any immediate symptoms of importance. Death is an exceptional termination of thyroidectomy—even in very young animals. Pulmonary tuberculosis is generally the cause of death in the cases that succumb after the operation. Experimental cretinism and thyroid cachexia are not necessarily consequences of simple thyroidectomy—even in very young animals. After thyroidectomy there is not only a relative but also an absolute increase of weight of the parathyroid glandules. Thyroidectomy is followed by an absolute increase of weight of the pituitary body. Rogowitch, Stieda and Hofmeister found that after the operation there was vacuolization of the cells and exaggerated colloid formation in this body.

2. Simultaneous thyroidectomy and parathyroidectomy is followed by primary symptoms some fifteen hours after the operation. Among others, there are convulsive and paralytic phenomena. There is also pupillary dilation, ptosis, salivation, hypothermia and polypnea. Death generally terminates the scene at an early date and *rigor mortis* sets in within some five minutes. The animals that survive present secondary symptoms later on. Among these symptoms are noted physical wasting that may be due to pulmonary tuberculosis excited by the operation. Loss of hair in patches and a diseased condition of the skin in these patches is also noticed. This diseased condition is not due to a parasite. The pituitary body was hypertrophied.

- 3 Thyroidectomy with subsequent extirpation of the parathyroid glandules is followed by symptoms similar to those described under the last heading.

TRANSLATIONS AND ABSTRACTS OF CURRENT LITERATURE.

Pathological Anatomy and Nature of Dementia Praecox.

—DR. KLIPPEL: The brain is composed of tissues of various differentiation and vulnerability. In certain mental diseases all the cerebral tissues are affected indiscriminately, involving the neuro-epithelial (neurones and neuroglia) as well as the vasculo-conjunctive elements (vessels and meninges); in other mental diseases, on the contrary, the neuro-epithelial elements alone are affected. From a pathogenic standpoint it is important to determine which of the tissues is affected in connection with a given disease. In the author's four cases of dementia praecox there was no reaction of the vasculo-conjunctive tissue: no leucocytosis of the blood in the cerebral vessels, no diapedesis, no hyperemia, no proliferation or degeneration of the vascular walls. In some circumscribed parts he found neuroglial proliferation in the vicinity of the vessels. This, however, was a reaction of the neuro-epithelial but not of the vasculo-conjunctive tissue.

The author attaches the utmost importance to this particular reaction in dementia praecox: the presence of lesions of the neuro-epithelial tissue exclusively shows that the pathogenic process, involving the most highly differentiated and therefore most fragile tissues only, must be slight in intensity but deeply rooted not only in the subject affected but also in his ascendants.

In other forms of dementia, on the contrary, the vascular walls and conjunctive tissue presented decided reaction. Thus, in the inflammatory general paralyzes vascular lesions are noticeable and diapedesis is present. In the degenerative general paralyzes there is no diapedesis, but the vascular walls are noticeably involved. In senile dementia there is also vascular degeneration. In other dementias without paralytic syndromes and in simple, non-paralytic dementia of alcoholic nature there is also vascular degeneration.

All dementias are characterized by organic lesions, but in the so-called psychic dementia the lesion is limited to the neuro-epithelial elements. Some cases of dementia praecox have been

reported in which neuroglia proliferation was found around the vessels; but neuroglia is of epithelial, not conjunctive nature. If cases have been reported in which diapedesis was found, it is a question whether this was not due to some intercurrent infectious disease. The author did not find any leucocytosis in the cerebro-spinal fluid taken from living subjects affected with dementia precox. In dementia precox the brain may sometimes present minimum vascular involvement under the conditions mentioned above, whereas in the other dementias the vasculo-conjunctive tissues are invariably involved. This characteristic of dementia precox puts it quite apart from other dementias (*Revue de Psychiatrie*, October, 1904).

Syndrome of Brown-Sequard of Reflex Nature.—PROF. E. SCIAMANNA: Six cases are described and are divided into two groups. In one group it may be admitted, by a stretch of imagination, that the slight wound had penetrated into the spinal canal and involved the spinal cord directly. In the second group the supposition of direct involvement of the spinal cord is out of question. The cases of the first group present a striking analogy to Brown-Sequard's syndrome. The cases of the second group, while presenting a clinical picture of Brown-Sequard's syndrome, had the striking characteristics of having this syndrome in an inverted form: in all of the latter three cases the symptoms were particularly marked, but the disturbances of sensibility were on the side of the traumatism, while the paralytic symptoms were on the opposite side. Without giving here the special details of the cases, their gross feature are reported below. Case I, wound inflicted in the vicinity of the first two dorsal spinous processes, slightly to the left of the median line, was immediately followed by motor and sensory paralysis of the right lower limb, hypoaesthesia of the left leg and by a zone of anesthesia of the right mammary region. Case II, wound inflicted in the region of the left spinous process of the scapula and was immediately followed by paresis of the right upper limb, paralysis of the right lower limb, paresis of the right eye-lid, complete anesthesia of the left side of the body and of the inner and anterior surfaces of both upper limbs. Case III, lesion in the region of the left spinous process of the scapula, paresis of the right upper and lower limbs and of the left hand with anesthesia of the left side of the body, beginning with the second rib and of considerable areas of both upper limbs, hyper-

esthesia of the upper part of the chest. Case IV, lesion in the right clavicular region followed by paresis and hypertonia of the left limbs, hypoaesthesia of the left side of the body, etc. Case V, lesion in the region of the left scapula in its upper part, paresis of the left lower limb, left hydrarthrosis, hypoaesthesia of the right side of the body. Case VI, lesion in the middle line of the neck and above elbow of the left arm; paresis of the right upper and lower limbs, thermic and dolorific hypoaesthesia of the right upper limb, tactile hypoaesthesia of the right upper limb.

An ingenious explanation, based on the minute anatomy of the spinal cord, is given in regard to the second group of cases (*Annali dell'Istituto Psichiatrico della R. Università di Roma*, Vol. III, fasc. I, 1904).

On the Recent Conceptions of Hysteria and Suggestion in Their Relation to an Endemic of Demoniactal Possession.

—DR. UGO CERLETTI: A peculiar endemic of "demoniactal possessions" prevails in Russia, in the Department of Archangel, along the mouth of the river Petchora. The disease is known under the name of *Ikota*, the term meaning hiccoughs. The most characteristic traits of the affection, however, are principally polymorphous convulsive attacks. The striking feature of the disease is that it affects married women almost to the exclusion of all other persons, the newly married women, on the very day of their nuptials being the most susceptible victims. It is rarely that a married woman can be found there who had not been victim of this affection. Young boys and girls are immune from the affection and only rarely are cases found among old women. It is exceptional to find a man affected with the disease. The time at which the young women are mostly stricken with the disease is either a short while after marriage or on the very day of the wedding. Superstition coupled with great ignorance of the people accounts for this peculiar manifestation. Sorcerers are believed there to have the power of transmitting any ailment, their power being particularly potent in the matter of transmitting the *Ikota* to young women on the very day of their nuptials. With this belief firmly rooted in their minds the young women, proceeding homeward from the wedding ceremony, are apt to have an attack of the *Ikota* at the slightest provocation: the sight of another woman in a fit of convulsions, hearing some fatal words pronounced, inhaling the smoke of a cigarette, etc. Some subjects present preliminary disturbances before the onset of the convul-

sive attack, having spells of vertigo, feeling dizzy, having a sense of oppression about the heart and epigastrium, etc. The more severe cases shriek out loudly and then fall in convulsive fits.

A few chapters are devoted to the consideration of the psychology of the disease, the leading works on suggestion, hypnotism, endemics and epidemics of neuroses being considered. It is concluded that fear and expectant attention caused by superstition are the two main elements in the causation of suggestibility in the newly married women. Any insignificant exciting cause can then bring about the attack (*Annali dell'Istituto Psichiatrico della R. Università di Roma*, Vol. III, fasc. I, 1904).

On the Chemical Changes in the Epileptic. — DRS. GUIDI AND GUERRI: The cases selected for the study were those of essential epilepsy. The diet was uniform during the time of experimentation, consisting of 2,500 litres of milk, 600 grams of bread, for the men, and 400 grams for the women, 150 grams of vegetables. No other food was allowed the patients. Before the experiments began, each patient was made to become accustomed to this diet during the preceding ten days. The urine was examined for every twenty-four hours and its entire amount was scrupulously gathered. Besides the total amount and specific weight, the urine was examined for the following elements: the total amount of nitrogen, urea, uric acid, ammonia, total amount of phosphoric acid, phosphoric acid combined with earthy and alkaline elements, chloride of sodium and sulphuric acid.

The amount of nitrogen discharged is below the normal. Other investigators have found similar results.

Those who admit auto-intoxication to be the cause of epilepsy say that during the period of calm there is a slowing in the process of tissue changes and that there is an increase of the same after the attacks. The amount of urea excreted is, according to some, below the normal during the period of calm and above the normal after the attacks.

The authors do not agree with these opinions either as regards the urea or the uric acid. The amount of urea is pretty nearly within the limits of the normal, or perhaps a trifle less, during the period of calm, and does not tend to increase after the attacks. Nor is the excretion of uric acid excessive, and the interesting point is that there is no relation between the amount of uric acid excreted and the attacks.

The most interesting point in this research is that an augmented amount of ammonia excreted coincides with a decreased amount of urea excreted, and this is synchronous with the time of an attack. This, according to the authors, indicates a deviation in the catabolic process expressed by an impaired succession of transformation of carbonate of ammonia into urea; this impaired process probably contributes to the formation in the organism of a substance, the composition of which is similar to that of urea. The uric acid, however, is not the index of acid intoxication of the epileptic; it is the ammonia compounds excreted that constitute the index of this acid intoxication of the epileptic.

The opinions of others regarding the individual biological processes leading to the formation of urea and uric acid respectively are confirmed.

The total amount of phosphoric acid excreted is augmented during periods of repeated attacks, particularly where these are numerous. The authors disagree with Agostini regarding the amount of earthy phosphoric acid excreted after the attacks. This combined acid is always below the normal after the attacks. No definite relation, however, can be drawn between the attacks and the amount of this combined acid excreted.

The elimination of chloride of sodium was repeatedly found to be below the normal. This is in accordance with results of others. No definite relation, however, can be established between the attacks and this decrease. It may be that the retention of chloride of sodium is a protective function intended to prevent disturbance of osmosis in the tissues.

The amount of sulphuric acid eliminated is above the normal. Considered in connection with the augmented amount of phosphoric acid, there seems to be an indication that phosphorus and sulphur are greatly used up in the metabolism of the epileptic. This fact may have some bearing on the decreased resistance of the tissues containing these two elements to a large extent.

There is an inverse ratio between the elimination of urea and the ammonia compounds. The catabolic processes are impaired and there is an increased elimination of phosphoric and sulphuric acids (*Annali dell'Istituto Psichiatrico della R. Università di Roma*, Vol. III, fasc. I, 1904).

Further Notes on Glycosuria in Insanity.—DR. W. R. DAWSON: Twelve cases were examined and all presented

a preponderance of mental depression. The same was true of a series of cases previously reported. Eight of the twelve cases here reported were those of melancholia, two had chronic mania and were somewhat demented, and two were considerably demented. In all the cases the glycosuria may probably be put down as alimentary and due to the disorder of metabolism in the insane: the power of assimilating sugar is markedly diminished in melancholic and demented states. In two cases a certain amount of mental depression may have been dependent on the glycoemia, but on the whole the conclusion seems to be justified that provided glycosuria is transient it possesses no particular significance. Without attaching any particular importance to the fact, it is stated that in nine of the cases the glycosuria appeared or commenced in the months of May and June, and in the tenth a second appearance took place in May (*Transactions, Royal Academy of Medicine, Ireland, Vol. XXII, 1904*).

The Periodic Menstrual Oscillation of the Temperature Pulse and Respiration of the Insane During Menstrual and Amenorrheal Life.—DR. ALFREDO SALERNI: The maximum vital energy, temperature, pulse and respiration of normal woman is expressed one or two days before the onset of the menstrual flow; there is an abrupt decrease of this energy below the normal after the onset of the flow and a gradual increase to the normal towards the end of the flow. The same is practically true of the insane. The cases examined were those of dementia precox, hebephrenic type, paranoia and maniacal excitation. The maniacal excitation during the flow did not influence the temperature. In four cases the temperature was almost febrile—38.9 to 39.2 degrees C. Psychomotor reactions of various degrees are noticed before, during and after the flow. Some cases with amenorrhea present analogous reactions during the periods when the flow should presumably have taken place. These patients, however, were young subjects. Cases of amenorrhea of a few years' duration after the establishment of the menopause do not present the periodicity of psycho-physical reaction recorded above. The menstrual flow, therefore, before the period of menopause, is only one of the many expressions of periodic activity (*Rivista Sperimentale di Freniatria, Vol. XXX, fasc. II-III, 1904*).

Superalimentation with Sugar.—DR. HENRI DAMAYE: Alimentation with sugar has given good results in the Vill-Juif Asylum. The dose given daily is between 150 and 500 grams. Feeding with sugar is particularly beneficial in cases of wasting during the course of the various psychoses, as in maniacal excitation, sitophobia, etc. One patient who was fed artificially with three litres of milk per day increased in weight from 51 to 55 kilogs after she had been given in addition 300 grams of sugar per day during a period of three weeks. Many other similar cases are reported. It is suggested that sugar feeding be used in cases of neurasthenia, hysteria, tuberculosis and in other affections characterized by tissue waste. There are no draw-backs to this treatment. Some patients occasionally show gastric intolerance or constipation, that are readily corrected by suspension of the sugar for a few days. Hyperglycemia is seldom caused, but it is well to examine the urine every week. In Dr. Toulouse's wards special breads containing a given excess of sugar are baked for the epileptics (*Revue de Psychiatrie*, October, 1904).

Mourly Vold's Researches in Visual Hallucinations During Dreams and the Wakeful State.—N. VASCHIDE: A critical review is made of Dr. Vold's conceptions of hallucinations and some of the conclusions are as follows:

1. Hallucinations during dreams and the wakeful state may be the results of excitations other than visual and they may especially be caused by cutaneo-motor excitations.

2. Cutaneo-motor sensations play a most important part in the formation of visual hallucinations during dreams and the wakeful state respectively.

3. These hallucinations are the results not exclusively of cutaneo-motor excitations; there is also collaboration of the visual apparatus, the retina and the eye muscles. The actual participation of the visual apparatus is of secondary importance.

4. There is an intimate relation between hallucinations as they are manifested during dreams, the wakeful state and those characterizing various psychopathic disturbances. Cutaneo-motor sensations play an equally important part in all these cases (*Revue de Psychiatrie*, October, 1904).

From the Journal of Mental Science, October, 1904:

1. A Case of Double Consciousness. — DR. ALBERT WILSON: The patient was a girl, 22 years of age. She suffered from some affection that may have been meningitis, but this is not known positively. She then developed acute maniacal excitation. During this illness the patient presented some remarkable changes of personality—such as are seen in hysteria. The author does not term the curious manifestations by this name, but describes some nine or more different varieties of personality manifested by the patient that are quite interesting. Thus, the patient had spells of blindness, deafness, paralyses, etc. The most interesting of these manifestations, because the least easy to explain, was the patient's ability to draw the full figure of a nurse, etc., during her spell of blindness. To convince himself that this was the actual case, the author held a book between the patient's eyes and the sheet of paper on which she was drawing, or put his hands before her eyes while she was drawing—without in any way interfering with her drawing. Her sense of touch was highly sensitive during that period; she could detect a line drawn across her drawing paper, dates written at the top of her drawing paper merely by touching the paper where the intruding lines had been drawn. Plates of the drawings are given. During her normal state the patient could never draw, either before or after the illness.

2. The Educational Treatment of Young Epileptics. — DR. G. L. SHUTTLEWORTH: In 1899, an Act of Parliament provided for the examination by the School Board of all the epileptic children of school age. In London, 470 epileptic children were selected and classified for instruction in special classes at school or for treatment and education in special institutions—according to the cases. A list of institutions for the medico-pedagogic treatment of epileptic children in England is given and the merits of each remarked upon.

A Case of Progressive Paralysis Infanto Juvenilis Simulating Cerebello-Spinal Atrophy. Anatomopathological and Clinical Study. — DR. GAETANO PERUSINI: A detailed study of the case is presented, adding valuable material to the list of such cases (*Annali dell'Istituto Psichiatrico della R. Università di Roma*, Vol. III, fasc. 1, 1904).

Influence of Alcoholism on the Procreative Power and the Descendants. Experimental Research.—

DR. CARLO CENI: 1. Chronic alcoholic intoxication has a direct effect on the reproductive organs and lessens the fecundity. 2. Chronic alcoholism of the parents causes a diminished resistance in the offspring, accompanied by various developmental anomalies. This is brought about by direct action of the alcohol on the ovule and spermatozoa before conception takes place. 3. This diseased condition of the elements of reproduction favors arrest of development and anomalies of development during embryonic life at the slightest provocation. 4. Alcoholism of the parents is expressed in the offspring either by general debility in its various forms and precocious death or secondary partial arrest of development during embryonic life (*Rivista Sperimentale di Freniatria*, Vol. XXX, fasc. II-III, 1904).

Clinico-Anatomical Contribution to the Study of Huntington's Chorea.—

DR. CESARE ROSSI: All cases of Huntington's chorea present profound psychic impairment. Heredity and psychopathia are the two characteristic features of the affection. Families affected with this disease always present psychoses with the chorea, but some of the members may be affected with psychoses without having chorea. The true coefficient of the disease is not the chorea but the mental invalidity. Huntington's disease is essentially a psychosis of hereditary nature and may or may not be accompanied by choreic or epileptic convulsions. The degeneration of the nervous elements in this disease is primary and parenchymatous (*Rivista Sperimentale di Freniatria*, Vol. XXX, fasc. II-III, 1904).

A Fatal Case of Myxedema.—DR. HENRY C. DRURY: The patient was forty-two years of age and had had ten children. Although her appearance was not typical in every respect, the aspect of the skin and the infiltrated substance under it were characteristic. A remarkable feature was the temperature that ranged between 85 and 86 degrees C. Thyroid treatment was given, but the patient had been brought for treatment too late in the course of the disease and she died in delirious coma (*Transactions Royal Academy of Medicine, Ireland*, Vol. XXII, 1904).

The Effect of Pituitary Feeding on Nitrogenous Metabolism.—DR. HENRY M. JOHNSON: The pituitary substance used was obtained from young calves, and the experiments were made on dogs. The total nitrogen in one gram of pituitary substance was 0.114 grams. This increased amount of nitrogen ingested was far too small to account for the markedly increased elimination of nitrogen. The nitrogenous metabolism is considerably stimulated by the pituitary substance. The total amount of nitrogen, urea and phosphates in the urine is increased. The weight of the animal falls. Nitrogenous equilibrium does not return at once after the pituitary substance has been omitted from the diet (*Transactions, Royal Academy of Medicine, Ireland*, Vol. XXII, 1904).

The Limit Between Thermic and Dolorific Sensibility.—DR. G. NARDELLI: Experiments were made on various subjects, including normal, paranoiac, melancholic, paralytic dements and hemiplegiacs. In all cases it was found that there were two distinct senses for the perception of heat and of cold. The existence of a special dolorific apparatus and sense was not confirmed (*Annali dell'Istituto Psichiatrico della R. Università di Roma*, Vol. III, fasc. I).

Contribution to the Treatment of Epilepsy with Ceni's Method.—DR. GIOVANNI TIENGO: The results obtained were not uniform in all cases. Some patients improved considerably, while in others it was necessary to discontinue the treatment. The majority of the cases, however, were considerably benefited, and the author considers this treatment of value.—(*Rivista Sperimentale di Freniatria*, Vol. XXX, fasc. II-III, 1904).

BOOK REVIEWS.

Le Rôle Du Sel En Pathologie.—DR. CH. ACHARD, Agrégé, Faculty of Medicine, Paris, Physician to the Tenon Hospital. Monographies Cliniques No. 39. Masson and Co., Paris. The amount of chloride of sodium differs with the tissues. In

the plasma of the blood the salt is found in the form of chloride of sodium, while in the blood cells it is in the form of chloride of potassium. The lymph is comparatively richer in chloride of sodium than the blood as a whole. The cerebro-spinal fluid also contains a large proportion of the salt: according to Achard 5.25 to 6.75 grams per 1,000; according to Widal and Sicard, 5.40 to 7.20 grams per 1,000, and according to Nobecourt and R. Voisin, 7 to 8 grams per 1,000. In the tissues the salt is less abundant than in the fluids. The liver contains 1.982 grams per 1,000; the kidneys, 2.714 per 1,000; the muscles, 1.549, and the brain, 2.119 grams per 1,000. The total amount of chloride of sodium is more marked in younger animals than in the adult. Normally, the salt is excreted mainly with the urine. Under normal conditions the amount of salt remains constant in the respective tissues. Hyperchloruration and hypochloruration may be induced by special alimentation. By virtue of its properties chloride of sodium maintains the equilibrium of osmotic pressure and has a powerful influence on arterial pressure: a large amount of chlorides increases arterial pressure. Chloride of sodium has a protective influence on the tissues, its abundance decreasing the toxicity of poisons; it also lessens the therapeutic effect of certain drugs. This is not a specific virtue of common salt; sugar and urea have the same effect, although common salt accomplishes the work more readily. The abundance and the smallness of the molecules of salt, its diffusibility and solubility render it the most serviceable element in maintaining the physico-chemical equilibrium of the various tissues. In pathological conditions the chloride of sodium accumulates in the tissues, not in the blood. As there is an affinity between salt and water, an excess of salt in the tissues is soon followed by corresponding hydration of the latter. This process is followed by augmented body weight even before decided edema sets in. In chronic affections such an augmentation is of ominous meaning: in uremic and cardiac cases the retention of the chloride of sodium is followed by pulmonary edema; the cerebral symptoms of acute nephritis are due to cerebral edema (abundance of the cerebro-spinal fluid) as well as to other factors.

Many important details in regard to the study of chloride of sodium in the human organism are given under instructive headings, analyzing the rôle of salt in the normal and pathological conditions. Considering the importance given to-day to serotherapy in psychiatry, this monograph comes to us quite opportunely.

BOOKS AND PAMPHLETS RECEIVED.

Dr. P. B. Gannoutchkine.—Acute Paranoia. A Clinical Study. Lissner & Geshel, Moscow, 1904.

Dr. Ch. Achard.—Rôle du sel en pathologie. Monographie clinique, No. 39. Masson Co., 1904.

Transactions of the Royal Academy of Medicine in Ireland, Vol. XXII., 1904.

Dr. Ugo Cerletti.—Sulle recenti concezioni dell'isteria e della suggestione a proposito di una epidemia di possessione demoniaca. Roma.

Dr. Ugo Cerletti.—Sulla corteccia cerebrale dei vecchi. Ricerche. Roma, 1904.

Prof. Sante De Sanctis.—Pensare e conoscersi. Roma, 1904.

Prof. Sante De Sanctis.—Le probleme de la conscience dans la psychologie moderne.

Dr. Charles H. Hughes.—The Louisiana Purchase Exposition, the Neurasthenic and the Brain-tired.

Dr. Arthur Donaggio.—Il reticolo fibrillare endocellulare e il cilindrase della cellula nervosa dei vertebrati e metodi vari di colorazione elettiva del reticolo endocellulare e del reticolo periferico basati sull'azione della piridina sul tessuto nervoso. Con 5 tavole e 4 figure. Reggio Emilia, 1904.

Dr. Grace Peckham Murray.—Rectal Constipation in Women.

Dr. Biaute.—Rapport sur le service medical du quartier d'Aliénés de l'hospice général de Nantes pendant l'année, 1903.

Dr. Grace Peckham Murray.—The Prevention of Uterine Disease.

Dr. Sergio Sergi.—Il solco di Rolando ed il lobo frontale nell'*Hylobates Syndactylus*. Con due figure.

A Discussion of the Vital Statistics of the Twelfth Census. Bulletin 15.

Report of the Superintendent of the Indian Schools, 1904.

Nineteenth Annual Report of the Adirondack Cottage Sanitarium, Saranac Lake, 1903.

Drs. Antonio d'Ormeo and Ferdinando Maggiotto.—Ricerche sul ricambio materiale nei dementi precoci. Seconda nota. Ricerche urologiche; fosfati, solfati, cloruri, urea, acido urico. Ferrara, 1904.

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TWO CASES OF FAMILIAL HEREDO-SPINAL ATROPHY (FRIEDREICH'S TYPE) WITH ONE AUTOPSY, AND ONE CASE OF SO-CALLED ABORTIVE FORM OF FRIEDREICH'S DISEASE.

ANATOMOPATHOLOGICAL AND CLINICAL STUDY.

BY PROF. G. MINGAZZINI, *Royal University of Rome, Italy,*
and

DR. G. PERUSINI, *Rome, Italy.*

(*From the School of Neuropathology, Rome, Italy.*)

(*Concluded.*)

IV.—THE SO-CALLED ABORTIVE FORM OF FRIED- REICH'S DISEASE.

We have seen above that the forms of Friedreich's disease could not be considered within any strict lines of demarkation. We have demonstrated that between the Friedreich and Marie type of the disease there existed many intermediary forms and we have touched on the fact relating to the difficulty of diagnosing with accuracy these forms of the disease. Besides all forms considered up to the present, there exists still another that is designated as an abortive form. The diagnosis of this form of the disease is accompanied by more difficulties than is that of its other forms. In connection with this form of the disease we shall consider the synopsis of the main traits of the affection of our patient, B. X., as given below.

1. Dubious familial nature of the affection.
2. The onset of the disease was subsequent to some acute febrile disease. There was weakness of the limbs.
3. Nystagmiform movements.
4. Slight scoliosis, cerebello-ataxic gait and static ataxia.
5. Impaired patellar reflexes.
6. Special senses and speech intact.
7. Slight mental defect.

The characteristics in favor of the diagnosis of Friedreich's disease are: the familial nature (?) of the affection, nystagmus, scoliosis, nature of the gait and the static ataxia. On the other hand, the onset following a febrile disease, the pains, the integrity of speech, the almost unimpaired patellar reflexes and the absence of any marked deformity of the feet do not, in themselves, invalidate the diagnosis of the disease.

Abortive forms of the disease are always difficult to diagnose, and Friedreich's disease makes no exception to the rule. Every author has experienced this difficulty in diagnosing Friedreich's disease. Raymond, for instance, goes so far as to say that when all other characteristic symptoms of the disease are absent, the diagnosis may be based on the sole presence of ataxi-cerebellar gait. Brissaud admits that typical cases of Friedreich's disease are rare, and he presented to his students a case to illustrate Friedreich's disease that did not have scoliosis, nystagmus, disturbances of speech or deformity of the feet. When the diagnostic strong-point is reduced to the simple disturbance of gait, one is necessarily confronted with the Charcot-Raymond tripod, of which such a gait is also characteristic, namely, *sclérose en plaques*, tabes dorsalis and cerebellar ataxia. And recently, Pastrovich made a diagnosis of Friedreich's disease in a case, eighteen years of age, in whom the disease set in at the age of sixteen years. The guiding points in the diagnosis were: cerebello-ataxic gait, Babinski's sign, slow and progressive course of the disease, normal patellar reflexes, absence of heredity, pupils, sensibility, bladder and bowels normal and absence of pains. Pastrovich bases his diagnosis on the strength of an analogous case reported by Senator. The latter author, speaking of his case, says that it presents the "purest form of Friedreich's disease, free from the accessory signs that set in later on in the course of the disease." Criticising this view, Schultze ironically remarks that Senator has discovered the "chemically pure preparation of Friedreich's disease." Leaving criticism aside, our patient was unable to hold herself on her feet unless supported by an attendant. When in the upright posture, she tried to keep her balance by widening her basis of support. When attempting to walk she tottered, stumbling to the right or to the left. These conditions sufficiently illustrate the type of the disease. Whether the familial nature of the disease exists or not, the diagnosis of Friedreich's disease is not invalidated.

If we consider what has been enumerated, the deformity that commenced about the left foot, the hyperextension of the large toe and the particular attitude of the hand and comparing this with the case of Cestan and Sicard, we could, without hesitation, pronounce

our case as one of Friedreich's disease. We are confronted with many difficulties, however, in making a diagnosis of such a case. Thus, some symptoms that are absent in the beginning of the disease may set in later on. The symptoms, on the contrary, that are present at the onset of the disease are not always easy of interpretation. One is obliged, under such circumstances, to be guided by preconceived diagnostic conceptions. Such a condition confronts us in our case. We are reduced to base our diagnosis principally on the character of the gait and another symptom of delicate interpretation—the nystagmiform movements. The deformity of the feet and the slight scoliosis may always be questioned here as to the diagnostic value. Under the conditions presented, however, it seems reasonable to speak of the presence of Friedreich's disease in a stage of evolution. On the other hand, it seems quite doubtful whether the case is simply one of cerebellar affection. We have been severe in criticising our own case and in comparing it to that published by dePastrovich. The negative characteristics of our case were quite similar to those found in dePastrovich's, and the presence of disturbances of subjective and objective sensibility in these patients is too well appreciated to-day to be regarded in the light of Friedreich's original opinion. In his own case, dePastrovich tries to differentiate it from *tabes juvenilis*, *sclérose en plaques* and ataxic paraplegia of Gowers. DePastrovich says that his case is not one of heredo-cerebellar ataxia of Marie's type on account of the precocious onset, the absence of alteration of the pupils, the optic and oculomotor nerves, although in Marie's type a tardy onset is not an absolute condition and although the Brissaud-Londe type is not characterized by any disturbances of the visual apparatus. We do not understand why dePastrovich did not consider his case as one of simple cerebellar origin when the only symptom present was cerebellar gait. We shall try and consider how much he has trespassed the legitimate clinical limits.

Thus, cerebellar disturbances are expressed by: impaired equilibrium of motility, in contrast to comparatively intact muscular force and sensibility, muscular asthenia, nystagmiform movements, slight tremors of the upper limbs, a variable condition of the deep reflexes and disturbances of speech that are not always present.

We think that our case may be considered within the limits of the above syndrome. Apart from all other considerations, the picture of cerebellar disturbances given above excludes, *per se*, *tabes juvenilis* and *sclérose en plaques*. It remains to consider cerebellar lesions or those spinal lesions that may be confounded with cerebellar atrophy. There is nothing that points to the presence

of a tumor. The only possibilities that remain are cerebellar sclerosis and atrophy. We shall not enter into any discussion regarding acquired cerebellar atrophies. Those familiar with the question will admit that the clinical criteria in hand do not lend themselves to the differential diagnosis in question. As regards our case, if we were certain of the familial character of the disease, it would be an important diagnostic trait—but only from an anatomical point of view. Clinically, however, acquired cerebellar atrophy and the more or less systematized heredo-familial cerebellar atrophy do not differ. The fact that the disease in our case set in subsequently to an acute infectious disease speaks neither in favor of an acquired cerebellar atrophy nor in favor of heredo-cerebellar atrophy. An infectious disease only acts as an exciting agent on a predisposed nervous system, and, broadly speaking, we agree with Adler's theory in this sense of the meaning. The disease that interests us should certainly not be considered as an acute cerebellar syndrome or as any other form of acute curable ataxia, as described by Bechterew, Chnitzer, Concetti and others. In his differential diagnoses, de Buch has even brought into the field the question of hysteria. This latter need hardly be considered in our case. The rather rapid development of the disease contrasts with the rather slow development usual in Friedreich's disease and Brissaud-Londe's type of it. This development may suggest the idea of its being an acquired disease. The question then arises whether there are any criteria by which an acquired cerebellar atrophy could be distinguished clinically from a familial cerebellar atrophy. The answer to this question is negative. Londe's attempts in this direction have not been crowned with success. Loew remarks that these attempts are more specious than decisive. To summarize, it seems to us that not only is it difficult to distinguish between Marie's and Friedreich's type, but the two are confounded with acquired cerebellar atrophy when the symptomatology is reduced to simple cerebello-spinal ataxia.

V.—ANATOMY, PATHOLOGICAL PHYSIOLOGY AND PATHOGENESIS.

We shall not take up the question of the anatomical pathology of Friedreich's disease. We only wish to classify our data and make a few remarks regarding them. The material on which the anatomopathology of this disease is based is rather scant, being sustained by the following studies: three of the four cases reported by Friedreich; the sixth case reported by Friedreich and Schultze, one by Schultze, one by Newton-Pitt, two by Rutimeyer, one by Letulle and Vaquez, one by Blocq and Marinesco, one by Guizzetti, one by Mirto, one by Burr, one by Dana, one by Simon and Philippe, one by Bonnus, one by Clarke, one by Auscher, one by Kahler and Pick, one by Brousse, one by Everett Smith, one by Erlicki and Rybalkin, two by Vincelet, one by Dumon, one by Richardson, two by Philippe and Oberthuer, one by Mackay, one by Greenless and Purvie, one by Rennie, one by Tedeschi and one by Rossi. The latter cannot at all be discussed. We shall not examine every case individually. Some of them that were published at an early date were not well studied and are incomplete in relation to the present knowledge, and some are doubtful as to their nature. Indeed, Auscher's case is considered by Guizzetti and Vincelet as unauthentic, although Dumon considers the criticism of the case insufficient. Among the other cases criticised by Vincelet are those of Kahler and Pick, Brousse, Everett Smith, Erlicki and Rybalkin. On the other hand, Thomas and Roux criticise Vincelet's latest case, saying that its anatomopathology is not that of Friedreich's disease. We pass in silence the much-discussed cases of Menzel and Meyer, considered by many as an intermediary type between those spoken of above.

Returning to our case, its macroscopic characteristics do not differ from those found in other cases. Thus, the brain was normal, the spinal meninges were thickened, and the cerebellum, that is the main point of discussion in the various theories, was normal in our case, as it was in the majority of the other cases. Transverse sections of the spinal cord made at different heights showed a special grayish coloring of the degenerated posterior columns.

Besides the question regarding the cerebellum, opinions differ also in regard to the topography of the lesions in the various cases and the possibility of establishing a type. Thus, Vincelet believes that the anatomopathology of Friedreich's disease does not present any definite type. For although there are some constant lesions, there are others that are inconstant. The constant lesions generally found are: sclerosis of Goll's column, quite variable sclerosis of Burdach's column, atrophy of the posterior horns and roots, and degeneration of Lissauer's zone. Among the inconstant lesions are: degeneration of the direct cerebellar tract, of Gower's column, of the anterior columns and horn and alterations of the ependymal canal. Dumon, on the contrary, is more sanguine in his opinion and thinks, on the ground of the study of twenty-two cases, that it is possible to bring to light the anatomopathology that corresponds to the items enumerated in Soca's synopsis. He presents this corresponding anatomopathology as follows:

1. *Constant Lesions*: sclerosis of Goll's and Burdach's columns, Goll's column always being more involved.

2. *Almost Constant Lesions*: sclerosis of the direct cerebellar tract. Sclerosis of the crossed pyramidal tract and of Gower's column. Ependymal alterations varying with each individual case. Cerebellar lesions are generally absent.

3. *Less Frequent Lesions*: sclerosis of the direct pyramidal tract. Atrophy of the anterior horns. Lesions of the spinal ganglia and of the peripheral nerves. Lesions of the meninges.

These apparently different conclusions are essentially identical. The difference is that of form rather than of substance.

As has been remarked above, Marie says that every patient has Friedreich's disease in his own way. Vincelet goes a step farther when saying that the anatomopathology varies with the symptomatology of every case. It seems to us, however, that with more cases in hand than we now possess it would be possible to take a broader view of the subject.

We have demonstrated that clinically our case corresponds to the type described as Friedreich's type and, to be more precise, to the form called integral. It remains to be seen whether our

microscopic findings correspond to those that are generally accepted.

It seems to us that there is a correspondence between the anatomical findings. Thus, there was sclerosis of Goll's column that was more marked than that of Burdach's, atrophy of the posterior horns and roots and degeneration of the zone of Lissauer. In fact, in our case, the sclerosis of Goll's column is most apparent and can be followed up to the medulla oblongata. The sclerosis of Burdach's column is less marked and is essentially variable at different heights of the spinal cord. And we wish to remark with Mirto that this difference is noticeable not only at different heights, but even on sections taken from almost the same height. This marked variability in the topography of the lesions in our case seems to go hand in hand with the relative intactness of the posterior roots. It may be of interest to note that in the cases recently reported by Dumon, Philippe and Oberthuer (two cases) the lesions of the posterior roots correspond exactly with those of the posterior columns. The facts presented seem to warrant the conclusion that perhaps all the fibres of the posterior columns are of exogenous origin. As has been shown, indeed, besides the lesions by contiguity, there is no part of the posterior columns, judging from their comparative intactness, that can be said to be of endogenous origin. The only noticeable difference in the degree of lesion at various levels of the spinal cord is *in relation to the posterior roots*.

The cornu-commissural zone, where, according to many opinions, the endogenous fibres meet, is certainly not completely spared at any level of the spinal cord. The variability of its degree of lesion is similar to that found in the antero-median and posterior-radicular zones. In the cervical region only does the cornu-commissural zone appear unimpaired, or pretty nearly so, as compared with the condition of the roots. We make mention of this because in some cases reported by Bonnus, Guizzetti, Mackay, Rutimeyer, and others, mention is made of the more or less absolute integrity of the *centrum ovale* of Flechsig and of the cornu-commissural zone. It seems to us reasonable to repeat with Schmaus that Goll's column is degenerated in its entity, Burdach's column in its median part and an unimpaired zone is found along the internal limits of the horn. Leaving aside the discussion of lesion by contiguity, the conception of the involvement of the endogenous and exogenous fibres is out of question. We need hardly discuss Combes' statement that those fibres of the posterior columns which are spared in tabes are attacked in Friedreich's disease.

The alteration of Lissauer's zone is quite noticeable in our case

and seems to be accepted by all as a constant lesion. Rutimeyer and Newton Pitt are exceptions, perhaps, in the list of authors. At the most, the difference of opinion applies to the entity of the lesion and to its greater or lesser extension at different levels of the spinal cord. Blocq and Marinesco have found lesions at the level of the lumbar region only. Lesions of the posterior roots were constantly found, as was the case in our study. In this instance, also, the difference applies only to the degree of lesion. Unfortunately, it has been impossible for us to study the spinal ganglia. Blocq and Marinesco and Schultze found these ganglia intact or almost so. Mirto, on the contrary, found rarification of the nervous reticulum, slight connective tissue hyperplasia and conspicuous atrophic degeneration (pigmentary, granular, hyaline, vacuolated) of the ganglionic nervous elements. Guizzetti also speaks of cellular atrophy, and Mackay's findings were similar to Mirto's. Dumon found a congestive condition, vascular dilatation and brown pigmentary deposits. We have not followed out the study of the peripheral nerves. Examination of the roots of the cauda equina gave negative results, as they were absolutely normal. Mirto found partial degenerative neuritis of the peripheral motor nerves and integrity of the muscular and cutaneous nerve endings. Guizzetti found atrophy of the peripheral nerves, especially involving the sensory fibres. In his much discussed autopsy, Auschner found that fibres of ordinary calibre were accompanied by many fine fibres and nervous tubes deprived of myeline. This made him suppose that the lesion was of evolutionary nature. Bonnus also found degeneration of the peripheral nerves. In many cases, however, no mention is made of this fact. According to Clarke, these nerves remain intact.

The direct cerebellar tract is frequently affected. In our case, the lesion has followed the usual course, beginning at the lower dorsal region and attenuating in degree towards the medulla oblongata. We shall have occasion to again refer to this matter later on.

Opinions differ as regards the condition of Gower's column. Its degeneration is clearly indicated by many authors, such as Newton Pitt, Rutimeyer, Blocq and Marinesco, Mirto, Dana, Burr, Simon and Philippe and Bonnus. According to Marie, when the disease is sufficiently advanced, the lesion is constantly present. This lesion was very slight in Clarke's case and only partial in Guizzetti's case. In our case also the lesion extends beyond the direct cerebellar tract, but is not very much marked. According to Mirto and Giuffré, retrograde degeneration takes place by extension.

In our case the column of Türk was absolutely intact. The crossed pyramidal tract seemed to be attacked in the lumbar region, but the lesion becomes more and more attenuated as it nears the fifth and sixth dorsal roots. If it is noticeably higher, the degeneration is limited to a simple zone of rarification, asymmetrical on both sides and not easily appreciable. The lesion of the direct pyramidal tract has been repeatedly noted by Friedreich, Burr, Philippe, Bonnus and Mackay. The absence of symmetry of this degeneration on both sides so frequently met with has led some authors to suppose that there was an anomaly of decussation of these pyramids. It may be said that the degeneration of the crossed pyramidal tract exists as a rule. Although Marie thinks that the localization and the aspect of the fibres in the various degenerated segments (ascending) and the absence of clinical symptoms indicate that the fibres belong not to the pyramidal tracts, but to Gower's column and the direct cerebellar tract, his opinion is not accepted by all. Many admit that fibres of different nature run through the pyramidal tract. The lesion of the ascending pyramidal tract, however, is to-day sufficiently verified. On the other hand, the lesions of the column of Türk and the presence of Babinski's sign would indicate that the crossed pyramidal tract is also involved.

It is difficult to admit, Schmaus remarks, that the fibres of the pyramidal tract do not degenerate. Although degeneration of the pyramidal tract exists in our case, it can be affirmed with certainty only as regards the lumbar region.

As we all admit that the direct cerebellar tract has its origin in the column of Clarke, it seems that the columns of Clarke are continued in some groups of cells at the base of the posterior horns in the lumbar enlargement. We do not feel authorized to speak of a direct cerebellar tract in the lumbar enlargement, but it may be useful to recall that Rothmann claims that it exists even in the *conus terminalis*. This fact has a direct bearing on the existence of a lesion of the pyramidal tract. Thus, the direct cerebellar tract is quite accustomed to surprises, so to speak, and can be the seat of ascending or descending degeneration. Therefore, the criticism applied to the variability of the course of the pyramidal tracts cannot be too severe. The scrupulousness would be too extreme if one were to insist on the integrity of the crossed pyramidal tract in our case.

As regards the triangular tract of Helweg in our case, we are not certain as to the interpretation of its condition. We can follow this tract from the first cervical root to the bulbar olive. We cannot say positively, however, whether or not its condition is nor-

mal. If here is any degeneration, it is certainly of slight degree. Some of the fibres seem to be thin, but it is difficult to say whether or not the method of staining is responsible for this appearance. According to Obersteiner, this special appearance of the fibres of this column is due to the Pal method. While the thinness of these fibres has often been observed in pathological conditions, it is also frequently observed in normal conditions. Besides, the appearance of these fibres is quite variable, and Marburg does not seem to have happily represented this particular condition of the fibres in his latest atlas. In the last edition of his work, Bechtereff calls attention to the tardy myelinisation of the fibres of this column, that he calls periolivary, and reserves his opinion concerning the relation of the olivary cells and the column in question. In our case it is difficult to say how much of the degeneration is simply peripheral and how much represents the degeneration or rarification of the triangular tract of Helweg, properly speaking.

Cellular lesions of the columns of Clarke have been invariably found by various authors, excepting in Clarke's case. The lesion of Clarke's column was quite noticeable in our case, and it is hardly necessary to dilate here on its significance in relation to the degeneration of the direct cerebellar tract. In our case the degeneration in the dorsal and cervical regions was slight and did not correspond to the sclerotic condition of Clarke's column. It seems reasonable to suppose that the direct cerebellar tract has its origin in part in the posterior roots, as has been maintained by some Italian authors. The cellular elements of the anterior horns in our case seemed to be intact, and this seems to be the rule in the other reported cases. Friedreich and Rutimeyer's cases are exceptions to this rule. Vincelet suggests the idea that in these latter cases the cellular lesions of the anterior horns may have had some relation to the muscular atrophy that characterized the cases. In our case, the cellular integrity of the anterior horns was contrasted by atrophy and considerable disintegration and retraction of the cellular elements of the posterior horns, as was shown by their imperfect staining. In this respect, our observations correspond with those of others. Nor is the obliteration of the ependymal canal wanting in our case—the obliteration reaching to the level of the medulla. Among its constant alterations reported by various authors are: bifid conditions, ectopia and quite frequent total or partial obstruction. Peri-ependymal glioma has also been noticed by Déjerine.

The meninges in our case were quite thickened. This thickening was not more marked about the posterior columns, but seemed more accentuated in the lumbar region. It is difficult to say

whether the thickening was more marked about the entrance of the posterior roots. The question of the meninges in Friedreich's disease remains a disputable point. Thus, Newton Pitt, Blocq and Marinesco, Bonnus and Clarke have found them intact. Others have reported more or less marked lesions of the meninges.

The cerebellum in our case was perfectly normal histologically. In Clarke's case it was atrophied, but there was a tumor that had caused partial destruction of the substance. In the cases of Auscher and Erlicki and Rybalkin the cerebellum was small but normal.

In our case the cerebral cortex was absolutely normal.

We feel that the short analysis presented by us warrants our conclusion that our case was one of Friedreich's disease. The essential points presented were: sclerosis of the posterior columns, more marked in Goll's than in Burdach's column. Atrophy of the posterior horns and roots. Lesion of the direct cerebellar tract. Lesion of the crossed pyramidal tracts. Slight lesion of Gower's column. Obliteration of the ependymal canal. Integrity of the anterior horns and roots and absence of cerebral and cerebellar lesions.

The second part of the anatomopathology of Friedreich's disease concerns the nature of the lesions that characterize it. It seems to be convenient to consider the significance of the affected systems of fibres in connection with the physiopathology of the disease. Opinions are very much divided on this question. As our specimens were treated with Mueller's fluid it was impossible to obtain positive results. The van Gieson method, the carmine stain and the double stain as well as Mallory's method are useful in the study of the neuroglia because of the facility with which it stains in pathological conditions. It is more difficult, however, to judge of the various lesions without using more modern methods. While it is possible to obtain certain results with Weigert's method in specimens treated with Mueller's fluid, the finer traits that are looked for to-day cannot be obtained by this method. The difficulties in staining as applied to the neuroglia fibrils also apply to the cellular elements of the nerves and neuroglia. It is impossible to obtain a specific nuclear and protoplasmic stain in specimens treated with Mueller's fluid. A complete interpretation of the anatomopathological facts is therefore impossible.

In our case we have been able to find the special disposition of the tissues known as "tourbillons." The presence of this structure raises two questions: one regarding its specific significance and the other regarding its nature. This structure is considered by Déjerine and Letulle as pure neuroglia sclerosis characterized by

neuroglia proliferation, a special morphological appearance and almost absolute integrity of the blood vessels and meninges. This, according to them, contrasts with the neuroglia and interstitial sclerosis that are always characterized by involvement of the vessels and meninges. The "tourbillons" structure is found exclusively in the posterior columns, the sclerosis being purely neuroglial, in contrast to the common sclerosis of the antero-lateral cords. This seems to have a bearing on the supposition, they say, that Friedreich's disease depends on impaired development due to an anomaly of the ectoderm.

It does not seem opportune here either to criticise this theory or to discuss the glioses and scleroses in connection with the question in hand. According to Weigert, neuroglia proliferation is much more marked in *sclérose en plaques* than it is in Friedreich's disease. Déjerine, on the contrary, says, in his latest work on the disease of the spinal cord, that it is impossible to make any distinction between the sclerosis found in Friedreich's disease and that encountered in the various diseases of the spinal cord. He further agrees with Guizzetti that no specific significance attaches to the "tourbillons" and that it may be found in other affections than in Friedreich's disease. Further, the "tourbillons" are found not only in the posterior columns, but also in the other sclerosed columns. According to Marie, the "tourbillons" is a relic of an old sclerosis dating from the time of the early development of the nervous system. Déjerine objects to this opinion. Schmaus says that even when the neuroglia proliferation is intense and there is a combination of peri-ependymal gliosis with obliteration of the central canal the neuroglia proliferation should be considered as a secondary sclerosis. Even under these conditions it is not correct to draw any definite conclusions regarding the nature of the disease. Indeed, according to Schmaus, an abnormally small spinal cord may be due to another old sclerotic process. When, however, the medulla oblongata is synchronously decreased in size, it is reasonable to speak of impairment during the process of development. According to Guizzetti, Friedreich's disease is due to a congenital predisposition that favors a progressive atrophy of the cellular and fibrous elements, leaving the vascular system intact. We have already had occasion to express our opinion regarding Adler's idea on this question.

We have mentioned above the opposed views of Blocq, Marinesco and Marie as against those of Déjerine and Letulle. If on the one hand, however, the idea of pure neuroglia sclerosis is untenable, the supposition, on the other hand, of arrested development remains as a simple theoretical reasoning. To speak in more

concrete terms, we shall consider the condition of the meninges and the blood-vessels. In our case, there was decided thickening of the pia and of its septa. There were also large tufts of fibrils that seemed to be partially connective tissue, although it is impossible to affirm this positively. In the medullary tracts we found glial elements that seemed to denote a progressive process in the glial elements themselves. This presence and aspect, however, does not differ from that found in other morbid processes. In our case, it would seem that a slight peripheral degeneration of the spinal cord corresponded to the thickening of the pia. Nevertheless, we cannot say that there was always an absolute correspondence between the attenuation of the pial thickening and the degeneration of the spinal cord. The meningeal thickening and its septa have been especially mentioned in many cases by Friedreich, Friedreich and Schultze, Rutimeyer, Mackay, Dana, Dumon, Philippe and Oberthuer. In our case we found thickening of the vascular walls, and other authors have made similar observations. Newton Pitt even considered this thickening as the origin of the sclerosis. Blocq and Marinesco, Philippe and Oberthuer and Dumon speak of the vascular thickening.

We shall not enter into any discussion of the subject, but simply wish to repeat that vascular and meningeal alterations are found in a large number of cases.

Senator, taking up Hammond's idea, claimed that the clinical symptomatology and the anatomopathology of Friedreich's disease had its origin in a cerebellar lesion. That the lesion consisted of a simple arrest of development of the cerebellum. The absence of symptoms of irritability is explained by this fact. Senator sustains his view by the interpretation he gives to Menzel and Nonne's cases and Marchi's experiments. According to these experiments, destruction of the cerebellum causes degeneration of the antero-lateral columns, of the anterior part of the direct cerebellar tract and of a certain number of fibres of the pyramidal tract. In Menzel's case there was atrophy of the cerebellum with spinal lesions typical of Friedreich's disease. In Nonne's case there was atrophy of the cerebellum and spinal cord, but the sclerotic lesions of the spinal cord analyzed above were absent.

There is a double difficulty in the interpretation of the physiopathology of Friedreich's disease. These difficulties are due to the fact of our insufficient knowledge of the functions of the cerebellum and the functional significance of the fibres of the posterior column. The researches of Luciani, Thomas, Adler and others, regarding the function of the cerebellum, are sufficiently known to need any comment. All agree that cerebellar lesions are

followed by cerebellar ataxia. The difference of opinions applies to the details of the question. One of us has also published a contribution to this study, in which the direct and indirect relations between the cerebellum and the cutaneous sensory nerves, the nuclei of the oculo-motor nerves and the acoustic nerve are considered (*).

We have already considered the different opinions regarding Friedreich's disease, Marie's cerebellar heredo-ataxia and the various intermediary forms. We have seen that while heredity existed in some cases, it was wanting in others. That while the disease was properly termed familial heredoataxia in some cases, the term was invalid as applied to others. It seems to us that the term "familial nature" of the disease should be taken in the largest sense of the word, indicating that in a given family there is a special predisposition to this disease. Senator's views, cited above, according to which Friedreich's disease depends on a cerebellar lesion, has been both vigorously sustained and opposed. The contradiction is explained when it is seen that Friedreich's disease may exist, without there being any lesion of the cerebellum. In other words, the cerebellum may appear intact, although its function is impaired. Goret seems to elucidate somewhat this moot question. According to him, the constant lesions of Friedreich's disease involve two kinds of neurones: the peripheral sensory neurones and the initial neurones of the cerebellar centripetal paths. He accepts Marie's theory regarding the pyramidal tract. Explaining the marked lesion of the direct cerebellar tract and the slight lesion of Gower's column, he says that the elements affected are the centripetal protoneurones and the initial neurones of the centripetal cerebellar path, while the central neurones remain intact. Remaining on these premises, he explains the abolition of the tendon reflexes by lesions of the centripetal protoneurones. The incoordination of movements should not be ascribed, he says, to anatomical or hypothetic functional lesions of the cells of the cerebellar cortex itself but to lesions of the direct cerebellar tract. The lesions of the centripetal protoneurones are here not accompanied by pains and disturbances of the general sensibility, as is the case in tabes, because here symptoms due to compression are out of the question and the matter is reduced exclusively to impaired development. The absence of disturbances of the general sensibility is also due to the slowness of development of the disease, during which supplementary function is readily established.

* Mingazzini. *Indice Bibliographica*. See also Silvagni. Monograph on Vertigo.

Sensory impressions are conveyed, he says, by way of the centripetal protoneurones that contribute to the formation of the short and medium radicular fibres (cornu-radicular and external *bandelette*). These latter remain intact in Friedreich's disease, while they are attacked in tabes dorsalis. As so many of the centripetal neurones, that contribute to the formation of Goll's column, are affected, the disappearance of the collateral reflexes is a natural consequence. The impairment of Clarke's column, into the structure of which enter the longer radicular fibres, is also explained. Finally, the unimpaired muscular sense is explained by the integrity of the terminal protoplasmic ramifications of the centripetal neurones.

As for Marie's cerebellar heredoataxia, it is due to a systematic involvement of the cerebello-cerebral neurones or of the Purkinje cells. The persistence of the reflexes is due to an intact condition of the fibres of the posterior column. Exaggeration of the reflexes is due to the participation of the cerebello-spinal neurones. Disturbances of equilibrium of cerebellar origin is explained by lesions of Purkinje's cells. Speaking of the narrowing of the visual field, impaired vision and amblyopia, Brissaud says as follows: cerebellar atrophy is accompanied by impairment of the upper cerebellar peduncles. The condition of the latter is intimately related by continuity or contiguity with the nuclei of the oculo-motor nerves, the corpora quadrigemina and the external corpus geniculatum. Londe thinks that impaired visual function is caused as follows: the cerebellar optic fibres run from the anterior bigemina through the superior cerebellar peduncles and end in the cerebellar cortex. The function of these fibres is impaired when there is lesion or destruction of the Purkinje cells. Gerest says that the frequency of visual disturbances in Marie's type and their rarity in Friedreich's type is explained by a destruction of the Purkinje cells in Marie's type and by their integrity in Friedreich's type.

The frequency of trophic disturbances in Friedreich's type is explained by the spinal lesions similar to those found in tabes. The rarity of trophic disturbances in Marie's type is due to the integrity of the spinal cord.

SUMMARY.—Gerest thinks that hereditary ataxia is due to arrest of development of the centripetal neurones and especially of those constituting the secondary central path of Van Gehuchten. This path is formed by a superposition of three kinds of neurones, each of which can be attacked individually during the course of evolution. Lesions of the lower neurones (centripetal protoneurones

and cerebello-spinal neurones) are expressed clinically by Friedreich's disease. Lesions of the upper neurones (cerebello-cortical) are expressed clinically by cerebellar heredo-ataxia of Marie. Finally, in some cases, as in the instance of Menzel, the lesion may involve synchronously two kinds of neurones, causing clinical symptoms common to both affections. Under these circumstances characteristics common to both diseases are represented and this form of disease figures under the heading of hereditary ataxia.

However acceptable some of Gerest's views are, they cannot be all endorsed. Thus, impairment of the spinal ganglia, the peripheral nerves and the muscular sense may exist in Friedreich's type. Under these conditions, if we are not mistaken, the limitation of the disease to one part of the centripetal protoneurone is not acceptable. The objections are still more serious as regards the disturbances of vision. Indeed, the superior cerebellar peduncles are in relation with the red nucleus only. The latter, in its turn, is in relation with the ventral nucleus of the thalamus, that no one has ever considered as being in relation with the tractus. Although Marchi claimed to have seen degeneration of the fibres of the tractus after extirpation of the cerebellum, no one has ever confirmed his views. It is impossible to comprehend, therefore, in what manner the cerebellum can be in relation with the optic nerve and how a cerebellar lesion can be followed by degeneration or atrophy of the optic nerves. Up to the present time, anatomy has not yet shown us how there can be a direct relation between the nuclei of the oculo-motor nerves and the cerebellum.

As regards the trophic changes, many authors are inclined to believe that they are equally frequent in Friedreich's and Marie's type. This is particularly true of scoliosis.

Speaking of the physiopathology of the disease, Grasset said, in 1882, that Friedreich's disease was characterized by a myelitis due to a systematic sclerosis of the posterior columns, on the one hand, and to diffuse myelitis, on the other. Now, he concludes that Friedreich's disease is characterized by ataxi-cerebellar symptoms, comprising lesions of all the neurones and paths of orientation, the direct cortical tracts (posterior columns), the cerebellar or indirect tracts (direct cerebellar tract and Clarke's column). Lesions of the posterior cords explain the abolition of the patellar reflexes and the inco-ordination of movements. Lesions of the direct cerebellar tract explain the cerebellar symptomatology of the disease. The absence of Romberg's sign is explained as follows: lesions of the cerebellar neurones cause defective orientation even when the eyes are open. The control examination with the

eyes closed becomes valueless. If, however, as Grasset would have it, Romberg's sign indicates a lesion of the posterior columns, but integrity of the cerebellar neurones, and if the absence of Romberg's sign, while there is motor inco-ordination, indicates a lesion of the cerebellar neurones, combined or not with lesions of the posterior columns, it becomes difficult to accord these views with clinical facts. Thus, whenever Romberg's sign is spoken of in connection with Friedreich's disease, it would be necessary to suppose that the cerebellar neurones are not affected. Such a clinical supposition would certainly be gratuitous. On the other hand, it would be difficult to explain the existence of Romberg's sign during life and the finding of lesions of the cerebellar neurones after death. Such cases were reported by Friedreich and Schultze, Letulle and Vaquez, Blocq and Marinesco, Dumon and Philippe and Oberthuer. To these may be added Vincelet's much discussed second case.

Stcherbach presents a different interpretation of the clinical symptomatology of Friedreich's disease. He draws a parallel between the phenomena observed in the latter disease, cerebellar heredoataxia and tabes dorsalis. According to this author, a great many fibres of Goll's and Burdach's columns that run up to the cerebellum do so either directly or after having been interrupted in the corresponding nuclei. The posterior cerebellar fibres and the cerebellar sensory fibres come together from the posterior roots. The existence of these posterior cerebellar fibres explains how a great part of the posterior columns may be degenerated without there being any disturbance of sensibility. Admitting that the cerebellum is the centre of co-ordinate movements, it is easy to explain Friedreich's disease without disturbances of sensibility in the beginning of the affection. The ataxic symptoms may take place when the posterior cerebellar fibres, the direct cerebellar tract and part of Gower's column are affected. Later on, the disturbances of speech are due to lesions of the fibres of the centripetal cerebello-cerebral axis. Asthenia and tottering gait are explained by lesions of the cerebellum itself. Finally, the intentional tremor and the choreiform movements are explained by lesions of the centrifugal cerebellar tracts. Hereditary cerebellar ataxia differs anatomically from Friedreich's disease. In hereditary cerebellar ataxia the cerebellar centres of co-ordination are directly affected without there being lesions of the centripetal cerebellar tracts. Friedreich's disease differs from tabes in so far that Friedreich's disease is due to a systematic affection of the entire cerebellar system, involving successfully all the neurones of the spinal cord, those of the cerebral axis and the cerebellum itself. Tabes

is due to a segmentary affection in which the protoneurons only are affected. Cerebellar symptoms are manifested in the latter part of the disease only, when the lesion spreads beyond Clarke's column.

The theory presented above is based on old data and is not supported by clinical facts. The symptomatology of familial ataxia does not develop with the regularity presented above. Bonardi justly remarked that the motor disturbances met with in tabetic ataxia are most complex in nature and most difficult to understand. His theory of the lesion of the functional cerebellar neurons, however, does not shed any light on the subject. Nor are Touche's recent studies of this subject convincing. Probst considers the cerebellum as a regulating apparatus of the muscular movements in voluntary, involuntary, automatic and reflex acts. He denies the existence of direct cerebellar centrifugal fibres in the *spinal cord*. The systematic affection of the spinal cord can hardly be accepted to-day. The notion of an initial vascular and meningeal lesion is becoming more and more accepted. And a very strict division of primary and secondary degeneration of the fibres is not accepted to-day. Pandy classifies Friedreich's disease with the centripetal ataxias. It seems to us that the degeneration of the fibres of the peripheral nerves found in this disease warrants its classification with the mixed ataxias (Déjerine). On the other hand, Rossolimo and Kojewnikoff think that the cerebellar lesions in heredo-cerebellar ataxia are not sufficiently marked to explain the clinical symptoms. They think, however, that there exist sufficiently marked lesions of the higher motor centres to explain the great symptomatic variety.

We do not propose to present any new theory regarding familial ataxia. We only wish to draw a parallel between the varied symptomatology and the pathology of the disease. Thus, in Fraser's case there was cerebellar atrophy. In Nonne's case there was integrity of the spinal cord. Nonne's case is nearer Friedreich's than Marie's type, but it did not present spinal ataxia. In Fraser's case the ataxia was increased when the eyes were shut and there were intentional choreiform movements. In Menzel's case there was cerebellar atrophy and sclerosis of the spinal cord, as is the case in Friedreich's type; in Menzel's case the ataxia is spinal. Vincelet considers that his second case, François Haudeb, was not a pure type of Friedreich's disease. Switalski completes the analysis of the case and Thomas and Roux show in this case and in the sister, Amelia Haudeb, that the lesions are identical. Thus, the cerebellum was smaller than normal, but the entire cerebro-spinal axis was smaller than normal. The only degeneration

seen was that of the direct cerebellar tract, the direct cerebellar tract in the restiform body and in the lateral nucleus of the medulla oblongata and, finally, in Gower's column. Meyer's case, that served to construct Marie's type, presented a doubtful cerebellar lesion, while the cerebello-spinal system was affected. Miura's two cases clinically represent Marie's type. The onset was tardy, at twenty-five and thirty-three years of age respectively, there was narrowing of the visual field, impaired vision and alteration of the papillæ. At the autopsy of one of the cases the cerebellum, Pons Varolii and the medulla oblongata were small, while the brain and spinal cord were intact.

We shall finally consider the two recent cases by Barker, or the 18th and 20th case of Sanger Brown, studied and illustrated in a manner so masterful that it calls forth the admiration of the European workers. We shall not analyze the clinical pictures of these cases. Anatomically, the following was found: the brain and spinal cord were relatively small. The spinal cord, the medulla oblongata and the Pons Varolii was in each case small in proportion to the cerebrum. The fissures of the cerebral cortex were in both cases well developed. The convolutions and sulci seemed normal. The study of the cellular topography with a view to establish the difference between the two cases is interesting.

Although diminished in size in both cases, the cerebellum was of normal configuration. The superior, middle and inferior cerebellar peduncles were all relatively small. The diminution in size, however, is not particularly marked. In each case the spinal cord was smaller than normal. In case 18 it was smaller than in case 20. In both cases there was marked degeneration of the white and gray substance of the spinal cord, the medulla oblongata and the cerebellum. The degeneration was more marked in case 18, the difference being that of degree rather than of nature. In both cases the elements involved were: the nervous cells the nervous fibres of the centripetal paths including one system of exogenous fibres of the posterior funiculus (apparently corresponding to the third fetal system of Trepinski), the dorsal nucleus of Clarke, the direct cerebellar tract of Flechsig in the lateral funiculus and in its continuation in the restiform body. In these structures the degeneration is extensive. The elements affected here were: the dentate nucleus of the cerebellum, *the brachium conjunctivum* and probably also the inferior olivary nucleus of the medulla oblongata. In the more advanced cases there was an insignificant diminution in the number of the cells of the anterior horns and of the fibres of the anterior nerve roots. An increased amount of the neuroglia tissue corresponded to the degeneration of the white

fibres in the spinal cord. This increased neuroglia tissue was relatively more marked in the dorsal funiculus than in the area of the lateral funiculus corresponding to the direct cerebellar tract. Comparing this case to Meyer's, the author finds that the lesions of the neurones are nearly identical in both instances. The difference of extent of the involvement corresponded to the clinical manifestations in the respective cases.

Unfortunately, the second part of the author's work, in which he proposes to treat of the relation between the anatomopathology and the symptomatology has not yet appeared.

Thomas and Roux do not accept the autonomy of Marie's syndrome and believe that its lines of demarkation are too narrow. The cerebellar system is always affected in the disease, but the lesion may involve various tracts. Thus, in Fraser's, Nonne's and Miura's cases there was a central involvement. In Menzel's case there was central, cerebellar and spinal involvement. In Meyer's case there was a marked predisposition in the cerebellar tracts in the spinal cord. Thomas and Roux say that while in Menzel's case there was cerebellar atrophy of a degenerative nature, the lesions found in Nonne and Miura's cases were quite different, and in Spiller's case, of familial nature, there was sclerotic atrophy. Hereditary cerebellar ataxia is considered as a syndrome common to some familial affections of the nervous system depending either on various lesions of the cerebellum, degenerative atrophy, sclerosis, or on lesions of the afferent or efferent tracts. The question suggests itself, then, why is Meyer's case not one of Friedreich's disease and how should Barker's cases be classified?

In view of all the difficulties that face us in regard to Friedreich's disease, on the one hand, and Marie's cerebellar heredo-ataxia, on the other, we ask the following question: does the degeneration of the spinal tracts precede or follow those of the cerebellum? Can both be affected synchronously? As yet we have no positive answer to these questions.

While awaiting Barker's promised publication, one of us is pursuing the clinical and anatomopathological study of cerebellar atrophy in man. Meanwhile, Professor Mingazzini would have us consider the cerebellar and spinal disturbances under the following headings:

1. Cerebellar atrophies: Heredo-familial (Marie's type).
Isolated.
2. Spinal atrophies: Heredo-familial (Friedreich's type).
Isolated.

3. Cerebello-spinal atrophies: Heredo-familial.
Isolated.

Returning to our case, Robert X., it seems that it may be classed as one of heredo-familial spinal atrophy of Friedreich's type. Maria X. enters into the same group. B. X. may probably be classed as an isolated form, but we do not know to which of the above mentioned groups she belongs.

From an anatomical point of view, Friedreich's disease is characterized by a combined sclerosis. According to Schmaus, time alone can help determine the true nature of the disease. "Otherwise, it can only be said," he says in part, "that ataxia is due to grave degeneration of the centripetal paths of the posterior cords, the cerebellar tracts and Gower's column. That nystagmus and impairment of speech are due to lesions of the medulla oblongata. The absence of patellar reflexes is caused by disease of the roots and the posterior columns. These lesions naturally explain the pains, disturbances of sensibility and bladder troubles that set in later on in the course of the disease. We do not know anything regarding the cause of scoliosis and the deformity of the feet."

Rome, Italy, May, 1904.

This work was finished when Bing's monograph came to our notice, treating of "Ersatztheorie" of Edinger, regarding Friedreich's disease. Bing applies to Friedreich's disease Edinger's theory of compensation. According to this theory the function of the hypoplastic parts of the spinal cord is the cause of the alteration of these parts.

The proofs of this paper were in our hands when we found that we had omitted Pic and Bonnamour's case (1) that appeared contemporaneously with our work. The case in question was again analyzed by Guenot (2) in his thesis that also came to our notice when the proofs of our work were being corrected. We shall not enter into any detailed consideration of these additional cases or of that published by Barjon and Cade. The latter is cited by us; macroscopic findings only accompany it. As regards the case of Pic, Bonnamour and Guenot, it is characterized by an absence of familial nature of the affection and its development subsequently to an infectious disease. The autopsy revealed softening of the right cerebral hemisphere (that was expressed during life by hemianesthesia, and perhaps also hemianopsia of the left side of the body), lesions of the spinal cord generally found in Friedreich's disease, with degeneration of the crossed pyramidal tracts. In this case Gower's tracts were not impaired and the integrity of the cornu-commissural zone and the centrum ovale of Flechsig was apparent. This statement about the case seems to us to be too decisive.

Guenot lays stress on the "bulbar syndrome" in Friedreich's disease. The case presented by him showed sclerosis of the twelfth, tenth and eighth pairs respectively in connection with the clinical symptoms during life expressed by disturbances of circulation, respiration, impaired hearing and speech. The histological examination was made according to old technical methods—Mueller's fluid being used.

1: *Nouvelle Iconographie de la Salpêtrière*, April, 1904.

2. Contribution à l'étude clinique, anatomo-pathologique et histologique de la maladie de Friedreich. *Thèse de Lyon*, No. 183, July 23, 1904.

Erratum:—Vol. VI, No. 3-4, Fig. 2.—"H, Triangle tract of Helweg" should read "H, Triangular tract of Helweg."—ED.

BIBLIOGRAPHY.

Acquaderni.—Alcune considerazioni sulla malattia di Friedreich, *Rivista Critica di Clinica Medica*, No. 2, p. 21, 1903.

Adler.—Ueber angeborene Kurzelebigkeit einzelnen Theile des Nervensystems, *Neurologisches Centralblatt*, p. 159, No. 4, 1901.

Adler.—Die Symptomatologie der Kleinhirn-Erkrankungen, Wiesbaden, 1899.

Afflek.—Two Cases of Friedreich's Ataxia, *Lancet*, p. 12, No. 31, 1898.

Allard and Monod.—Pied bot paralytique simulant le pied de Friedreich, *Société de Neurologie de Paris*, p. 281, *Revue Neurologique*, 1901.

Allen Starr.—Three Cases of Friedreich's Ataxia, p. 194. *The Journal of Nervous and Mental Diseases*, No. 3, 1898.

Amouroux.—Etiologie et pathogénie de la maladie de Friedreich, Thèse de Paris, 1899.

Auscher.—Sur un cas de maladie de Friedreich, *Bulletin de la Société de Biologie*, p. 470, 1890.

Auscher.—Sur un cas de maladie de Friedreich avec autopsie, *Archives de Physiologie Normale et Pathologique*, p. 340, 1893.

Badrian.—Beitrag zur Kasuistik der Friedreichschen Krankheit, Inaugural Dissertation, Berlin, 1898.

Ballet.—Traité de Pathologie Mentale, pp. 20, 40, Paris, 1903.

Barker.—Description of Brain and Spinal Cord in Hereditary Ataxia, *The Decennial Publications of the University of Chicago*, Vol. X, 1903.

Barjon and Cade.—Liquide céphalo-rachidien et méningite chronique dans un cas de maladie de Friedreich, *Comptes Rendu de la Société de Biologie*, p. 247, March 2, 1901.

Batten.—Friedreich's Disease and Mental Defect, *Brain*, 1898.

Baumel.—Cas de maladie de Friedreich, *Mercredi Médical*, p. 49, 1891.

Baumlin.—Ueber familiaere Erkrankungen des Nerven systems, *Deutsche Zeitschrift fuer Nervenheilkunde*, p. 265, XX, 1901.

Bayet.—Maladie de Friedreich et hérédo-syphilis, *Journal de Neurologie*, No. 8, 1902.

Bayet.—Cas de syphilis familiale. Maladie de Friedreich, *Revue pratique des maladies cutanés*, p. 391, 1903.

Bechterew.—Ueber acute cerebellar Ataxie, *Neurolog. Centralb.*, p. 435, No. 10, 1902.

Bechterew.—Ueber acut auftretenden Störungen der Motilität mit den Merkmalen cerebellarer Ataxie bei Alkoholikern, Neurol. Centralb., p. 834, No. 18, 1900.

Bechterew.—Ueber das Olivenbündel des Cervicallentheiles vom Rückenmark, Neurolog. Centralb., p. 433, No. 12, 1894.

Bechterew.—Les voies de conduction du cerveau et de la moelle, p. 164, Paris, 1900.

Berdez.—Un cas isolé de maladie de Friedreich, Revue Méd. de la Suisse Romande, p. 304, No. 6, 1896.

Bernabes.—Sur la maladie de Friedreich, Progrès Méd., p. 211, 1890.

Best.—Notes on a Case of Rapidly Fatal Glycosuria in a Case of Friedreich's Disease, Lancet, p. 371, Vol. 1, 1899.

Besold.—Klinische Beiträge zur Kenntniss der Friedreich'schen Krankheit (Hereditäre resp. juvenile Ataxie), Deutsche Zeitschrift f. Nervenheil., p. 157, V, 1894.

Bing.—Die Abnützung des Rückenmarks (Friedreich'schen Krankheit und Verwandtes), Deut. Zeit. f. Nervenh., Hft., 1-2, p. 163, 1904.

Biro.—Einige Mitteilungen ueber die Friedreich'schen Krankheit, Deut. Zeit. f. Nervenh., p. 164, Bd. 19, 1901.

Blocq.—Un cas de maladie de Friedreich, Arch. de Neur., p. 217, 1887.

Blocq and Marinesco.—Sur l'anatomie pathologique de la maladie de Friedreich, Arch. de Neurol., p. 331, 1890.

Bonardi.—Ataxie tabétique et titubation cérébelleuse selon la doctrine des neurones, Revue Neurologique, p. 1013, 1901.

Bonnus.—Un cas de maladie de Friedreich à début tardif, Nouvelle Icon. de la Salpêtrière, p. 178, t. XI, 1898.

Bonnus.—Contribution à l'étude de la maladie de Friedreich à début tardif, Thèse de Paris, 1898.

Bouchaud.—La maladie de Friedreich chez deux frères jumeaux, Jour. des Sc. Méd. de Lille, p. 265, No. 37, 1899.

Bourneville and Crouzon.—Un cas d'affection familiale à symptôme cérébro-spinaux. Diplegie spasmodique infantile et idiotie chez deux frères. Atrophie du cervelet, Progrès Méd., p. 273, No. 17, 1901.

Brissaud.—Leçons sur les maladies nerveuses, p. 45, 1893-4.

Brissaud and Londe.—Sur un cas d'héréditaire ataxie cérébelleuse, p. 129, Revue Neurol., 1894.

Brousse.—De l'ataxie héréditaire, Thèse de Montpellier, 1882.

Bramwell.—Remarks on Friedreich's Ataxia with Notes of Three Cases, Brit. Med. Jour., p. 896, Oct. 2, 1897.

Burr.—A Contribution to the Pathology of Friedreich's Ataxia, University Medical Magazine, Philadelphia, 1894.

Carenville.—Un cas de maladie de Friedreich, Revue méd. de la Suisse Romande, p. 208, No. 4, 1898.

Carslaw.—An isolated Case of Friedreich's Ataxia, ref. in Glasgow Medical Journal, p. 191, No. 54, 1900.

Caton.—Friedreich's Ataxia, British Med. Jour., p. 1270, I, 1901.

Cestan.—Le pied bot de la maladie de Friedreich, Bull. de la Société Anatom. de Paris, p. 736, 1898.

Cestan and Sicard.—La "main bote" dans la maladie de Friedreich, Revue Neurologique, p. 1118, 1903.

Cestan and Sicard.—Les analgésies viscérales dans la maladie de Friedreich, Soc. de Neurol. de Paris, Nov. 5, 1903, also Revue Neurol., 1903, p. 1117.

Charcot.—Maladie de Friedreich. Clinique, 1888, March 13, April 10, p. 175 and 238.

Chauffard.—Maladie de Friedreich avec attitudes athétoïdes, Sémaine méd., p. 409, No. 52, 1893.

Chnitzer.—Sur l'ataxie cérébelleuse aigue, Questions russes de médecins neuro-psych., p. 552, Vol. V, No. IV, 1900, and Revue Neurologique, p. 307, 1901.

Clarke.—A case of Friedreich's Disease or Hereditary Ataxia with Necropsy, British Med. Jour., p. 1294, 1894.

Clarke.—Hereditary Cerebellar Ataxy in Two Brothers, p. 1640, British Med. Jour., Nov. 22, 1902.

Cohn.—Zwei Faelle von Friedreich'scher Ataxie, Neurol. Centralb., p. 302, 1898.

Combe.—La maladie de Friedreich, Ann. de Méd. et Chir. infantile, No. 23, 1898.

Combemale and Ingelrans.—A l'appui de l'origine infectieuse de certains cas de maladie de Friedreich, Echo Méd. du Nord, p. 211, VIII, 1902.

Combes.—Maladie de Friedreich. Essai historique, anatomo-clinique et physiologique, Thèse de Montpellier, 1902.

Concetti.—Sopra un caso di atassia cerebellare post-tifica in un bambino, La Pediatria, p. 240, Vol. VI, No. 8, 1898.

Cousot.—Observations de maladie de Friedreich, Bull. de l'Académie R. de Méd. de Belgique, Oct., 1902.

Crouzon.—Des scléroses combinées de la moelle, Thèse de Paris, 1904.

Couche.—Etude sur la maladie de Friedreich, Thèse de Lyon, 1887.

Czylharz.—Friedreich's Ataxie, Centralb. f. Allgm. Pathologie, p. 731, 1900.

Dana.—Acute Spinal Ataxia (non tabetic) and Its Relation to other Forms of Acute Ataxia, New York Med. Jour., p. 667, April 20, 1901.

Dana.—A Case of Friedreich's Ataxia with Autopsy, Postgraduate, New York, p. 328, Vol. XI, 1896.

Debove.—Maladie de Friedreich, Gazette des Hopitaux, p. 1065, 1890.

DeBuck.—Syndrome cérébelleux, Journal de Neurologie, No. 23, 1901.

DeGrazia.—Contributi alle malattie nervose sistematiche eredofamigliari, Archivio Italiano di medicina interna, f. 20, 1899.

Déjerine.—L'hérédité dans les maladies du système nerveux, Thèse d'agrégation, 1886.

Déjerine.—Semeiologia del sistema nervoso, Trattato di patologia generale di Charcot-Bouchard, Vol. V, p. 231.

Déjerine.—Sur une forme particulière de la maladie de Friedreich avec atrophie musculaire et troubles de la sensibilité, Comptes Rendus de la Société de Biologie, p. 42, 1890.

Déjerine.—Sur les différences de l'état de la sensibilité dans la maladie de Friedreich et dans la maladie de Duchenne, *ibidem*, p. 120.

Déjerine.—Sur les causes probables de l'intégrité de la sensibilité dans la maladie de Friedreich, *ibidem*, p. 105.

Déjerine.—Note sur l'anatomie pathologique sur la maladie de Friedreich, *ibidem*, p. 479.

Déjerine and Egger.—Contribution à l'étude de la physiologie pathologique de l'incoordination motrice (ataxie d'origine périphérique et ataxie d'origine centrale), Revue Neurologique, p. 397, 1903.

Déjerine and Letulle.—Sur la nature de la sclérose des cordons postérieurs dans la maladie de Friedreich, Comptes Rendus de la Soc. de Biologie, p. 127, II, 1890, and Semaine Méd., p. 81, 1890.

Déjerine and Thomas.—L'atrophie olivo-ponto-cérébelleuse, Nouvelle Icon. de la Salpêtrière, p. 330, 1900.

Déjerine and Thomas.—Traité des maladies de la moelle épinière, p. 427, Paris, 1902.

Demoulin.—Absence du caractère familial et étiologie infectieuse dans certains cas de maladie de Friedreich, Thèse de Lille, 1902.

DePastovich.—Di un caso singolare di morbo del Friedreich (ataxia ereditaria), Rivista di Freniatria, Vol. XXIX, 1902, f. 1-2, p. 343.

Deschamps.—Du traitement électrique dans deux cas de maladie de Friedreich, Annales d'électrobiologie, p. 349, May 15, 1898.

Dumon.—Contribution à l'étude de l'anatomie pathologique de la maladie de Friedreich, Thèse de Lyon, 1902.

Edleston.—A Case of Friedreich's Ataxia, British Med. Jour., Nov. 22, 1902.

Egger.—Fall von Friedreich'scher Ataxie, Corresp. Blatt f. Schweiz. Aerzte, p. 597, No. 19, 1898.

Erb.—Ueber hereditaere Ataxie mit Krankenvorstellung, Wandersammlung, etc., zu Baden Baden, ref. in Neurol. Centralb., p. 378, 1890.

Erlicki and Rybalkine.—Zur Frage ueber die combinirte Systemerkrankungen des Rueckenmarks, Arch. f. Psych., p. 693, B. 17, 1887.

Esposito.—Sulle malattie mentali famigliari, Il manicomio, Vol. XIX., No. 2, 1903.

Everett Smith.—A Contribution to the Pathology of Friedreich's Ataxia, Boston Medical and Surgical Jour., 1885.

Ewald.—Vorstellung eines Falles von Friedreich'scher Krankheit, Gesellschaft der Charité Aerzte, June 28, 1894, Berlin. Klin. Wochensch., p. 746, No. 32, 1894.

Fraser.—Defect of Cerebellum Occurring in Brother and Sister, Glasgow Med. Jour., No. 1, 1880.

Friedreich.—Ueber statische Ataxie und ataktischen nystagmus, Arch. f. Psych., p. 235, Bd. 7, 1876.

Friedreich.—Ueber degenerative Atrophie der spinalen Hinterstraenge, Virch. Arch., p. 391, Vol. 26, 1863.

Friedreich.—Ueber Ataxie mit besonderer Beruecksichtigung der hereditaeren Formen, Virchow's Arch., p. 140, Vol. 70, 1877.

Friedenreich.—Et Tilfaelde af hereditaer Ataxie (Friedreich's Sygdom), Hosp. Tid., 1891, Ref. in Neurol. Centralb., p. 211, 1892.

Fuerstner.—Drei Faelle Friedreich'scher Krankheit, Ref. Deut. Med. Wochenschr., No. 26, 1898.

Gasne.—Localisations spinales de la syphilis héréditaire, p. 126, Paris, 1897.

Gerest.—Application de la théorie des neurones à l'étude des affections nerveuses systématiques. Essai de classification rationnelle, Thèse de Lyon, 1897.

Giacchetti.—Malattia di Friedreich ed eredoatassia cerebellare di Marie, Rivista Critica di Clinica Medica, p. 425, No. 27, 1903.

Gilles de la Tourette.—Pathogénie et traitement des pieds bots, Leçons de clinique thérapeutique sur les maladies du système nerveux, p. 481, 1898, Paris.

Gilles de la Tourette.—La marche dans les maladies du système nerveux étudié par la méthode des empreintes, p. 50, Paris, 1886.

Gilles de la Tourette, Blocq, Huet.—Cinq cas de maladie de Friedreich, Nouvelle Iconographie de la Salpêtrière, p. 45, 1888.

Glorieux.—Un cas de maladie de Friedreich, Jour. de Neur. et d'Hypnologie, No. 15, 1898.

Goldscheider.—Diagnostik der Krankheiten des Nervensystems, p. 211, Berlin, 1903.

Gowers.—Manuale delle malattie del sistema nervoso, Vol. I, p. 471, 1894.

Grasset.—Diagnostic des maladies de la moelle, p. 30, Paris, 1901.

Grasset.—Les maladies de l'orientation et de l'équilibre, p. 98, Paris, 1901.

Grasset et Rauzier.—Traité pratique des maladies nerveuses, Vol. I, p. 823, Paris, 1904.

Greenless and Purvis.—Friedreich's Ataxia, Brain, p. 135, Vol. I, 1900.

Guizzetti.—Le alterazioni dei nervi periferici e dei gangli spinali in un caso di malattia di Friedreich, Riforma Medica, pp. 140-141, June, 1893.

Guizzetti.—Contributo all'anatomia patologica della malattia di Friedreich, II Policlinico, p. 438, 1894.

Hallion.—Des déviations vertébrales néuropathiques, Nouvelle Iconog. de la Salpêtrière, p. 136, 1892.

Heveroeh.—Ataxie cérébelleuse héréditaire Ref., Revue Neur., p. 41, 1898.

Hodge.—Three Cases of Friedreich's Disease, All Presenting Marked Increase of the Knee-jerk, Boston Med. Jour., p. 1405, June 5, 1897.

Hoffman.—Ueber hereditaere Ataxie, Allgem. Zeitschr. f. Psych., Bd. 56, p. 598, 1899.

Howard Gladstone.—Friedreich's Ataxia with Knee-jerks and Ankle-clonus, Brain, Vol. 4, 1899.

Hunter.—Case of Friedreich's Disease with Atrophy of the Left Leg, Glasgow Med. Jour., Vol. 54, p. 375, 1900.

Jacoby.—Cerebellar Ataxia, Boston Med. Jour., p. 210, No. 9, 1897.

Joffroy.—Observation de la maladie de Friedreich, Bull. et memoires de la société des hôpitaux de Paris, p. 88, 1888.

Karplus.—Friedreich's Krankheit, Jahrbuch f. Psych., Bd. 20, p. 413, 1901.

Kassirer.—Ein Fall von Friedreich'scher Ataxie, Neurol. Centralb., p. 513, No. 11, 1897.

Katz.—Das Symptomenbild der Friedreich'schen Ataxie nach akuter Infektionskrankheit, Deutsche Med. Woch., No. 37, 1898, p. 587.

Klippel and Durante.—Contribution à l'étude des infections nerveuses familiales et héréditaires, Revue de Médecine, Oct., 1902, p. 745.

Koenig.—Cerebrale Diplegie der Kinder, Friedreich'sche Krankheit und multiple Sklerose, Berlin. Klin. Wochenschr., Aug. 19, 1895, p. 716.

Kopczynski.—Ueber hereditaere Ataxie (Friedreich'schen Krankheit) Mendel's Jahresbericht, p. 473, 1899.

Krause.—Cas de maladie de Friedreich, Ref., Revue Neurol., 1893, p. 541.

Kahler and Pick.—Ueber kombinirte Systemerkrankungen des Rueckenmarks, Arch. f. Psych., Bd. 8, 1878, p. 251.

Ladame.—La maladie de Friedreich, Revue Médicale de la Suisse Romande, 1889, p. 397.

Lannois and Pariot.—Les lésions histologiques de l'écorce dans les atrophies du cervelet, Nouvelle Iconographie de la Salpêtrière, No. 6, 1902, p. 513.

Legrain.—Sur un cas de l'hérédo-ataxie cérébelleuse, Hopital civil de Bougie, Maloine, Paris, 1900 (?)

Lenoble.—Etude clinique sur cinq cas d'hérédo-ataxie cérébelleuse observées dans la même famille, Arch. Provinciale de Médecine, Dec., 1899.

Lenoble and Aubineaux.—Deux cas de maladie nerveuse familiale intermédiaire entre l'hérédo-ataxie cérébelleuse et la maladie de Friedreich, Revue Neurol., 1901, p. 393.

Lepine.—Maladie de Friedreich avec propulsion en avant, Revue de Médecine, 1896, p. 271.

Letulle et Vaquez.—Un cas de maladie de Friedreich avec autopsie, Comptes Rendus de la Soc. de Biologie, Vol. II, 1890, p. 21.

Leube.—Specielle Diagnose der inneren Krankheiten, Bd. II, 1898, p. 120, Leipzig.

Loew.—L'atrophie olivo - ponto - cérébelleuse type Déjerine-Thomas, Thèse de Paris, 1903.

Londe.—Sur deux cas familiaux d'hérédo-ataxie cérébelleuse, Revue Neurol., 1894, p. 521.

Londe.—Maladies familiales du système nerveux. De l'hérédotaxie cérébelleuse, Thèse, Paris, 1895.

Lop.—Sur un cas de maladie de Friedreich, Revue de Médecine, 1893, p. 396.

Lorrain.—Contribution à l'étude de la paraplégie spasmodique familiale, Thèse, Paris, 1898.

Mackay.—Pathology of a Case of Friedreich's Disease, Brain, Vol. 21, 1898, p. 435.

Mackenzie.—A Case of Non-hereditary Friedreich's Disease, The American Jour. of the Medical Sciences, No. 264, 1894, p. 421.

Magnus.—Bidrag till lären om de hereditär-familiäre nervesygdomme, Norsk. Mag. f. Lägeidensk, H. R., 1899, p. 265, abstract in Mendel's Jahresbericht, p. 473.

Mannini.—Alcuni casi di atassia ereditaria o malattia di Friedreich, Riforma Medica, No. 7, 1903, p. 180.

Margulies.—Ein Beitrag zu den Uebergangsformen zwischen Friedreich's Ataxie und Hérédotaxie cérébelleuse von Marie, Inaug. Dissertation, Berlin, 1901.

Marie.—Leçons sur les maladies de la moelle, p. 381, Paris, 1892.

Marie.—Malattia di Friedreich, Trattato di medicina di Charcot e Bouchard, Vol. 6, p. 418.

Marie.—Sur l'hérédotaxie cérébelleuse, Semaine Méd., 1893, p. 444.

Martin.—Un cas de maladie de Friedreich, Le Nord Méd., No. 85, 1898, p. 91.

Melot de Beauregard.—Ueber einen Fall Friedreich'scher Krankheit mit Störungen der Sensibilität, Inaug. Dissert., Berlin, 1894.

Menzel.—Beitrag zur Kenntniss der hereditären Ataxie und Kleinhirnatrophie, Arch. f. Psych., Vol. 22, 1891, p. 160.

Meyer.—The Morbid Anatomy of a Case of Hereditary Ataxy, Brain, 1897, part 79, p. 276.

Mingazzini.—Experimentale und pathologisch-anatomische Untersuchungen ueber den Verlauf einiger Bahnen des Centralnervensystems, Monatschr. f. Psych. und Neurol., Bd. XV, H. 5, 1904, pp. 95, 110.

Mirto.—Atassia di Friedreich ed atassia volgare, Giorn. dell'associaz dei medici e dei naturalisti, Vol. 4, Nos. 3 and 4, 1893.

Mirto and Giuffré.—Malattia di Friedreich, Trattato italiano di patologia e terapia medica, Vol. II, p. III bis, p. 181.

Miura.—Ueber l'hérédotaxie cérébelleuse Marie's, Mittheil-

ungen des Med. Facultaet der Kaiserlichen Japan Universitaet zu Tokio, Bd. IV, No. 1, 1898, also Neurol. Centralb., 1889, p. 132.

Moebius.—Diagnostik der Nervenkrankheiten, p. 414, 1894, Leipzig.

Morselli.—Contributo alla terapia del morbo di Friedreich, Nuovo raccoglitore medico, Nos. 11-12, 1903.

Murray.—A Case of Friedreich's Ataxia, Lancet, Dec. 24, 1898.

Namach.—A Case of Friedreich's Ataxia, non-hereditary, Medical Record, Aug. 11, 1894.

Newton Pitt.—On a Case of Friedreich's Disease. Its Clinical History, with Post-mortem Appearances, Guy's Hosp. Reports, Vol. XXIX, 1887, p. 367.

Nolan.—Friedreich's Disease (Hereditary Ataxia) Associated with genitous Idiocy, Dublin Jour. of Med. Sc., No. 3, 1895, p. 815.

Nonne.—Ueber eine eigenthumliche familiaere Erkrankungsform des Centralnervensystems, Arch. f. Psych., Vol. XXII, 1891, p. 283.

Olenoff.—Essai sur l'hérédité dans la maladie de Friedreich, Thèse, Montpellier, 1903.

Oppenheim.—Trattato delle malattie nervose, traduzione De-Pastrovich, p. 219, Milan, 1903.

Ormerod.—Some Further Observations on Friedreich's Disease, Brain, p. 461, Vol. X, 1887-88.

Ottersbach.—Ein Fall von Friedreich'schen Krankheit, Inaug. Dissertation, 1897, Bonn.

Obersteiner.—Bemerkungen zur Helweg'schen Dreikantenbahn, Arb. aus dem Neur. Instit. an den Wiener Univers., p. 286, 1900.

Obersteiner.—Die Variationen in der Lagerung der Pyramidalbahnen, Arb. aus dem Neur. Instit. an der Wiener Univ., p. 417, 1902.

Oulmond and Ramond.—Maladie de Friedreich et hérédo-ataxie cérébelleuse, Mercredi médical, No. 9, 1895, and Revue Neurologique, No. 8, 1895, p. 238.

Pacheco.—Ataxie héréditaire de Friedreich, Revista del Hospital de Ninos, Buenos Ayres, p. 123, I semestre, 1898, and in Revue Neurol., p. 20, 1899.

Pandy.—Neuritis multiplex und Ataxie, Klin.-therap. Wochenschr., Nos. 42-44, 1900.

Paravicini.—Observation d'ataxie cérébello-spinale dans l'enfance, Corresp. Blt. f. Schweiz. Aerzte, No. 10, 1901, p. 305.

Patrick.—Hereditary Cerebellar Ataxia, Jour. of Nervous and Mental Dis., No. 3, 1902.

Pearce and Swan.—Friedreich's Disease with a Report of a Fatal Case, Philad. Med. Jour., No. 21, 1900, p. 1201.

Peck.—A Case of Friedreich's Ataxia, Jour. of Nervous and Mental Dis., p. 505, 1899.

Pelizoeus.—Ueber eine eigenthumlichen Form spastisches Laemung, etc., Arch. f. Psych., XVI, 1885, p. 698.

Perregaux.—Maladie de Friedreich, Médecine moderne, 1895, p. 70.

Pesker.—Un cas d'affection familiale à symptômes cérébro-spinaux, Thèse, Paris, 1900.

Petit.—Maladie de Friedreich après rougeole, Jour. de clinique infantil, June 30, 1898.

Philippe and Oberthuer.—Deux autopsies de maladie de Friedreich, Revue Neurologique, 1901, p. 971.

Potts.—A Case of Friedreich's Ataxia, Jour. Am. Med. Assoc., Vol. XXXII, No. 12, 1899.

Pribram.—Friedreich's Ataxie, Deut. Med. Wochenschr., 1902, p. 171.

Pritzche.—Fortgeschrittene Friedreich'sche Krankheit mit Idiotie bei zwei Geschwistern, Inaug. Dissert., Marburg, 1901.

Probst.—Zur Anatomie und Physiologie des Kleihirns, Arch. f. Psych., Bd. 35, H. 3, 1902, p. 692.

Prochazka.—Un cas de maladie de Friedreich, Abstract in Revue Neurologique, 1901, p. 1006.

Putnam.—Friedreich's Ataxia. Boston Med. and Surg. Jour., 1901, p. 185.

Pusateri.—Contributo allo studio della sclerosi cerebrale atrofica con osservazion sull' origine del tapetum e del fascio periolivare di Bechterew, Il Pisani, Vol. XXII, f. 2, 1901.

Ranklin.—Friedreich's Ataxy, Lancet, Jan. 18, 1902.

Rauzier.—La maladie de Friedreich et son traitement, Nouveau Montpellier Médical, Sep. 11, 1898, Vol. VII, No. 37.

Rauzier.—De la maladie de Friedreich, Montpellier Méd., 1893, p. 725.

Raymond.—Tabès juvénile et tabès héréditaire, Progrès Méd., No. 32, 1897.

Raymond.—Leçons sur les maladies du système nerveux, 1898, pp. 329-399.

Raymond.—Scléroses systématiques de la moelle, Paris, 1894.

Rennie.—A Case of Friedreich's Hereditary Ataxy with Necropsy, British Med. Jour., July 15, 1899, p. 129.

Ribel.—Contribution à l'étude de la maladie de Friedreich, Thèse, Paris, 1894.

Richardson.—The Lesions in the Cord from a Case of Friedreich's or Hereditary Ataxia, Jour. Boston Soc. Med. Sc., Vol. III, No. 1, Oct., 1898, p. 25.

Rossi.—Due casi singolari di morbo del Friedreich, Manicomio moderno, 1893, p. 238.

Rossolimo.—Trois cas d'ataxie cérébelleuse héréditaire dans la même famille, Nouvelle Iconographie de la Salpêtrière, No. 1, 1899, p. 22.

Rossolimo.—Ataxie cérébelleuse héréditaire, Revue Neurol, 1899, p. 348.

Rothmann.—Die sacrolumbale Kleinhirnseitenstrangbahn, Neur. Centralb., Nos. 1-2, 1900, pp. 16, 66.

Roussel.—Contribution à l'étude de l'étiologie de la scoliose, Thèse, Paris, 1903.

Royet and Collet.—Sur une lésion systématisée du cervelet et de ses dépendances bulbo-protuberantielles, Arch. de Neurologie, 1893, p. 353, No. 81.

Ruffinet.—Essai clinique sur les troubles oculaires dans la maladie de Friedreich, etc., Thèse, Paris, 1891.

Ruetimeyer.—Ueber hereditaere Ataxie. Ein Beitrag zur den primaeren combinirten Systemerkrankungen des Rueckenmarks, Virchow's Archiv., Bd. 110, 1887, p. 215.

Ruetimeyer.—Ueber hereditaere Ataxie, Virchow's Archiv, Vol. XCI, 1883, p. 106.

Sanger Brown.—On Hereditary Ataxy with a Series of Twenty-one Cases, Brain, Vol. LVIII, 1892.

Schmaus.—Vorlesungen ueber die pathologische Anatomie des Rueckenmarks, Wiesbaden, 1901, p. 347.

Schoenborn.—Mittheilungen zur Friedreich'schen Ataxie, Neurol. Centralb., No. 1, 1901, p. 10.

Schultze.—Die Pathologie und pathologische Anatomie der hereditaren Ataxie, Neurol. Centralb., No. 13, 1883, p. 290.

Schultze.—Ueber die Friedreich'schen Krankheit und aehnliche Krankheitsformen nebst Bemerkungen ueber nystagmusaehnliche Zuckungen bei Gesunden, Deut. Zeitschr. f. Nervenheilk., Vol. V, 1894, pp. 27, 103.

Schultze.—Erwiederung auf den zweiten Artikel von Senator ueber hereditaere Ataxie (Friedreich'sche Krankheit), Berl. Klin. Woch., No. 33, 1894, p. 761.

Schwarz.—Fall von Friedreich's Ataxia, Deut. Med. Woch., 1902, p. 304.

Seeligmüller.—Hereditaer Ataxie mit Nystagmus, Arch. f. Psych., t. X, p. 222.

Seiffer.—Ueber die Friedreich'schen Krankheit und ihre Trennung in eine spinale und cerebellare Form, Charité Annalen, XXVI, 1903.

Senator.—Ueber hereditaere Ataxie, Friedreich'sche Krankheit, Berlin. Klin. Woch., No. 21, 1893, p. 489.

Senator.—Ueber hereditaer Ataxie, Friedreich'sche, Krankheit, 2-d article, Berl. Klin. Woch., No. 28, 1894, p. 639.

Sicard.—Le liquide céphalo-rachidien, p. 178, 1902, Paris.

Silvagni.—Patogenesi e Semiologia della vertigine, p. 21, 1897, Rome.

Simon and Philippe.—Un cas de maladie de Friedreich avec autopsie, Progrès Médical, No. 36, 1897.

Smith-Traviers.—Case of Friedreich's Ataxy, British Medical Jour., 1901, p. 150.

Soca.—Etude clinique sur la maladie de Friedreich, Thèse de Paris, 1888.

Soca.—Un nouveau cas de maladie de Friedreich, Nouvelle Iconographie de la Salpêtrière, 1888.

Spiller.—Four cases of Cerebellar Disease, One Autopsy, with Reference to Cerebellar Hereditary Ataxia, Brain, part 76, 1896, p. 588.

Stcherbach.—Ueber die Kleinhirnhinterstrangbahn und ihre physiologische und pathologische Bedeutung, Neur. Centralb., No. 23, 1900, p. 1090.

Stein.—Ueber einen eigenartigen Fall von Friedreich'schen Ataxie combinirt mit athetose, Berl. Klin. Woch., No. 49, 1897.

Stein.—Kasuistischer Beitrag zur hereditaeren Ataxie, Prag. Med. Woch., No. 12, 1902.

Stern.—Ett fall af hereditär ataxi, abstract in Neur. Centralb., 1903, p. 38.

Switalski.—Sur l'anatomie pathologique de l'hérédo-ataxie cérébelleuse, Nouvelle Iconogr. de la Salpêtrière, No. 5, 1901, p. 373.

Szczypiorski.—Maladie de Friedreich accompagnée de troubles trophiques chez un imbecile épileptique, Ann. Médico-Psychologiques, June, 1892.

Symes.—Friedreich's Disease, Dublin Journal of Medical Sc., Feb., 1897.

Tedeschi.—Die Friedreich'sche Krankheit. Kritisch und pathologische anatomische Untersuchung, Beitrag zur pathol. Anat. und zur allgem. Pathol. von Ziegler, XX, 1, 1895.

Thalwitzer.—Ueber zwei im fruestem Kindesalter begonnene Faelle von Friedreich'scher Krankheit, Inaug. Dissert., Berlin, 1897.

Thomas.—Le cervelet. Thèse, Paris, 1897.

Thomas.—Atrophie du cervelet et sclérose en plaques, Revue Neurologique, No. 3, 1903.

Thomas.—Atrophie ponto-cérébelleuse, Arch. de Neurol., 1903, p. 199.

Thomas and Roux.—Sur une forme d'hérédo-ataxie cérébelleuse à propos d'une observation suivie d'autopsie, Revue de Médecine, 1901, p. 762.

Variot.—Cas de maladie de Friedreich, Journal de Méd. et pharmacol., Brussels, 1895, p. 33.

Veraguth.—Ueber Friedreich'sche Ataxie mit Krankenvorstellung, Corresp. Blatt f. Schweizer Aerzte, No. 6, 1900, p. 173.

Verhoogen.—Deux cas de maladie de Friedreich, Jour. de méd. et pharm., Brussels, 1895, p. 33.

Versiloff.—Recherches expérimentale sur la fonction cérébelleuse, Jour. Russe de Nevropathol. et de Psych., also Revue Neurologique, 1903, p. 1045.

Vincelet.—Etude sur l'anatomie pathologique de la maladie de Friedreich, Thèse, Paris, 1900.

Vires.—Diagnostic et traitement des maladies du système nerveux, Montpellier, 1902.

Vizioli.—La malattia di Friedreich, Giornali di Nevropatol., 1885.

Voelker.—A Case of Hereditary Ataxia, abstract in British Med. Jour., No. 2038, 1899, p. 145.

Weber.—Drei neue Faelle von Reiner Hereditärer Ataxie. Deut. Med. Woch., Sep. 26, 1901.

Weigert.—Zur pathologischen Anatomie des Neuroglia fasergerüst, Centralb. f. Allg. Pathol., 1890, p. 729.

Whyte.—Four Cases of Friedreich's Ataxia with a Critical Digest of Recent Literature on the Subject, Brain, part 81, 1898, p. 72.

Wickel.—Ein Fall von Friedreich'scher Krankheit, Muench. Med. Woch., No. 8, 1900, p. 249.

Zabloudowski.—Sur un cas d'ataxie de Friedreich. Traitement par le massage, Vratch, No. 34, 1896. Abstract in Revue Neurologique, 1896, p. 618.

REMARKS ON THREE CASES OF MORBID LYING.

BY DR. RAYMOND MEUNIER, *Paris.*

The question of lying has been very little studied from the strictly psychological point of view. The few reports found in periodicals treat mostly of the pedagogic and moral side of the question.* Yet cases of lying studied from the psychological point of view would be most serviceable. The cases I am about to relate are above all well substantiated and the remarks I shall make are intended to indicate a possible orientation in the question. The three cases to be related below were studied in a well-known primary school in Paris. The directrice of the school lent me her personal aid by carefully recording in exact terms the morbid manifestation of lying of the three children. She personally made written reports of the incidents that interested me, and I subsequently studied the children myself. The histories given below were written out by the directrice of the school.

Case I.—A girl of the middle grade neglected her studies. When reprimanded, she said, crying: "I assure you, Madam, that I can do no better at present. I am so busy. My father is suffering with his eyes" (her father was an engraver), "my mother has to take him to the oculist every day and I have to attend to the cooking." The directrice consoled the child and often inquired how her father was. One day the little girl tearfully replied to one of these inquiries: "My father has become blind and there is no more hope for him." A fortnight later, the directrice met the mother of the child and expressed her feelings of sympathy. The mother was greatly astonished at this. An explanation brought out the fact that the child had fabricated every bit of the story of her father's illness in order to have an excuse for the dereliction in her studies.

* Duprat. *Le mensonge: étude de psychologie pathologique et normale*, Vol. I., No. 12. Alcan, Paris, 1903.

Case II.—R., eleven years of age, came to school, crying because, she said, her mother had died the previous night. Her father, she added, alarmed at the sudden onset of her mother's illness, had run about all over town for a physician but could find none. When one physician finally responded to the call and came to the house, it was too late—the patient was dead. The funeral was to take place within two or three days so that all the relatives should have time enough to reach the city on the date appointed for the funeral. The school teacher repeated this sorrowful story to her colleagues, one of whom said: "You must be mistaken about this child; she lost her mother when she was my pupil three months ago." An inquiry brought to light the fact that the child had fabricated the story for the second time. Her mother was living and healthy. The author then examined the child himself. She was eleven years of age, of medium stature and fat. Her physiognomy was calm, thoughtful and she looked one straight in the face when addressed. She was analytical towards her surroundings and herself. There was nothing of note about her parents—who led a regular family life.

Case III.—The directrice of the school presented to the children a project of a society called *Société de Vestiaire Scolaire*. The aim of this society is to provide clothing for needy school children. The directrice, therefore, made an appeal for a collection to be sent to the above society. One girl then stood up and said: "Madame, my uncle is cashier at the X., and has already gotten one hundred subscriptions among the people of that house. In a few days he will bring you the full list of subscribers and the money he has collected. During the two months that followed the child gave various excuses for her uncle's non-appearance. On questioning the child's mother, the directrice found that the child had concocted the story about the uncle: there was no uncle or any other relative connected with the business house she had mentioned. The author's examination of the child: she is thirteen years old, rather large for her age, anemic, excitable, talking rapidly, smiling as she speaks and presenting some disturbances in her speaking voice. Her movements are rapid, awkward and incoordinate. To all appearances, she seems to be a candidate for hysteria within a few years from now. Her mother is nervous and the father is not known.

The three cases reported above are those of morbid lying. Normal lying could be distinguished from its morbid form: a normal lie is generally told with some purpose in view and is more of

interest to moralists and sociologists than to psychologists. The three cases of morbid lying related above are cases of systematized deliriums: the development of such delirium annihilates, so to speak, the entire personality of the subject and his entire mental life is invaded by abnormal extra- and introspection. In the first case it may be supposed that the child lied for some purpose, but in the majority of such cases the utilitarian purpose is far overstepped: the delirium commands and systematizes all acquired impressions. There is a veritable splitting of the personality, in which the new "I" is developed at the expense of the normal "I"—that now appears only at certain intervals. The absence of utilitarian purpose is particularly apparent in the second case presented here. The calm physiognomy of the little girl and her pensive eyes reminded one of a sister of charity. In her case there was absolutely no desire to attain anything by declaring her mother dead twice during the course of three months. No doubt, the child had often presented to herself mentally the death of her mother, the despair of her father, who came with the physician—too late to render any assistance, etc., etc. The child even developed this trend of thought in every detail—following out the circumstances of the funeral, etc. Apparently her mind was overfilled with this imaginary mental burden of grief and she no longer could bear it alone—when she found relief in relating it to her elders. This she did in proper form—making her sad declaration with a flood of tears. Her entire mentality was absorbed in this delirium, and she repeated the story twice within three months in identical terms and expressions.

Each of the three cases related has its originality. The first case almost resembles normal lying, but the purpose for which the lie was fabricated makes it abnormal: the personality was sufficiently absorbed by the systematized delirium to reveal the morbid nature of the lying. The second case is typical of morbid lying: the lying was done without any purpose in view and resembled an obsession in every respect. The third case is less typical, but is interesting from the standpoint of the study of psychology of characters.

Without entering into any consideration of the value of such studies, I wish to remark that it is useful to study morbid lying from the psychological point of view—in its relation to delirious systematization of ideas. Such studies are of importance in pedagogy and sociology.

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ATTEMPT ON THE LIFE OF DR. VALLON.

While making his morning visit in his wards at Ste.-Anne, Paris, on October 4, 1904, Dr. Vallon was attacked by one of his patients but miraculously escaped being killed by him. The patient is suffering from delirium of persecution and previous to his admission to the hospital had attempted the life of a person whom he imagined to be his enemy. The medico-legal report of Dr. Vallon saved the patient from the hands of the law and he was sent to be cared for in Dr. Vallon's wards. The patient immediately included the physician among his imaginary enemies and systematically laid out a plan for taking his life. He managed to obtain a pointed knife in the kitchen and kept it in concealment for several months—with the intent of ending Dr. Vallon's life with it. On the date mentioned above the patient considered that the opportune moment for the execution of his design had arrived. Springing at Dr. Vallon as he was leaving the ward, the patient plunged the knife into his neck with such rapidity that neither the attendants nor the two internes who were near him could interfere in time. Dr. Vallon fell uncon-

scious. He was immediately transported to the surgical pavilion, where, fortunately, the surgeon of the Asylums of the Seine, Dr. Picqué, happened to be present.

A penetrating wound between two vertebræ was found and it was feared that the spinal cord had been cut, particularly because the accident was immediately followed by complete right hemiplegia. A few days later, however, Dr. Vallon could move his right leg and arm.

Dr. Picqué's report of the case is given in the *Annales Médico-Psychologiques*, November-December, 1904, as follows below.

The traumatism was inflicted with a long, narrow and dirty blade of a knife that had penetrated at a level of a transverse line passing horizontally at the level of the point of the mastoid process and two centimètres back of it. The wound was inflicted at a moment when Dr. Vallon's head inclined forwards. The blade cut a wound $1\frac{1}{2}$ centimètres wide and, according to Dr. Ballet, reached the spinal cord quite near the medulla oblongata. The loss of consciousness lasted twenty seconds. On regaining consciousness Dr. Vallon thought that he had received a blow on the head, but soon realized the true seat of the wound. From the moment he fell there was complete hemiplegia with hyperesthesia of the same side and anesthesia on the opposite side (Brown-Séquard's syndrome). Dr. Picqué saw Dr. Vallon about twenty-five seconds after the accident and immediately cleansed and dressed the wound antiseptically. Considering the responsibility involved, he abstained from exploring the wound until Professor Berger had responded to his call for consultation. On the following day chlorethyl was applied locally, the wound thoroughly cleansed, gauze drainage introduced that was changed twice and the wound healed without there being any elevation of temperature.

Dr Ballet stated on the first day that there had been no hemisection of the spinal cord and that the lesion was limited to superficial scratching of some tracts of Gower's column. He also said that he had had a case that was quite similar to the one in question and that complete recovery had taken place. As Dr. Vallon presented marked improvement within a few days after the accident, the prognosis is considered most favorable.

We express our sincerest wishes for Dr. Vallon's speedy recovery.

THE PERILS OF THE PSYCHIATRIST.

Few people realize that the physician who handles the insane is always exposed to danger of losing his life at the hands of this or that patient under his care. Every physician handling the insane is familiar with the common fact that patients suffering from delirium of persecution readily include their physicians among their imaginary enemies, the chief physician of a given service or hospital who treats them generally being the preferred victim. We have, in our own country, had quite a number of such victims. The latest victim abroad is Dr. Vallon, of Ste.-Anne, Paris. We give in this issue a detailed account of the circumstances under which Dr. Vallon nearly lost his life at the hands of an insane patient. Considering the ever present danger of this kind to physicians in charge of hospitals for the insane, the subject should be looked into by all concerned. Practically speaking, it is the attendant who is responsible for the patients' concealing instruments with which bodily harm can be inflicted. Attendants who have not had sufficiently convincing experience regarding the wisdom of *never* trusting an insane person, are apt to become lax in the supervision of the patients entrusted to their care. The results of the least opportunity for insane patients to carry off and conceal about their persons knives, hammers and other instruments, hardly need be depicted here. The case of Dr. Vallon is typical of them all. It seems practical to us that the histories of cases like that of Dr. Vallon be collected either in a separate volume or appended to the text-books of attendants and that the attendants be required to have a knowledge of such cases. A vivid picture of dramatic incidents of this kind is bound to produce some impression on the attendants' minds and to make them feel more deeply the responsibility they assume when undertaking to care for the insane. After all, the mere fact that physicians make the study of the insane a life work is no reason why the danger attending this work should not be minimized by all concerned.

ALCOHOLISM IN SCANDINAVIA.

We have on many occasions considered the question of alcoholism,—its destructive effects on the individual, the family and nations. We have elsewhere considered the question of prophylaxis against the spread of alcoholism and the evils consequent thereon and have pointed out that the most practical measure for preventing alcoholism was individual education. Our colleague, Dr. Legrain, confirms our views: in his report of his impressions

on the question of alcoholism, published in the *Archives de neurologie*, November, 1904, he considers the system of Government monopoly of drinking houses in Sweden and Norway and says that this system is not proof against alcoholism in these countries. The preventive measure, he says, of selling not less than a quart bottle of any alcoholic drink, has proven a failure. The subject purchasing a bottle of liquor is not allowed to drink it in the shop. The consequence is that either the alcohol is consumed at the home of the individual and the whole family becomes intoxicated or else the individual intoxicates himself on the street far more than he would under ordinary circumstances. It is a common thing, therefore, to find on the streets individuals intoxicated to insensibility. Besides, there are many so-called eating houses in which food is presumably sold, but the main feature of which is a cupboard well stocked with alcoholic drinks, to which every "guest" has free access. If the "guest" is caught in the act by an officer of the law, the proprietor declares that his guest was "stealing." The proprietor cannot be prosecuted because every citizen has the privilege of having his private supply of drinkables stolen. The monopoly in Russia has produced similar effects. Dr. Legrain says: instead of drinking within the enclosure of four walls, the people are found drinking and drunk on the streets. Commenting on these conditions, Dr. Toulouse also says that the desired reform can be attained only through the reform of the individual himself.

INTERNATIONAL CONGRESS OF PSYCHOLOGY.—This congress will be held in Rome, April 26-30, 1905, under the presidency of Prof. Luciani. Prof. Sergi will be vice-president, Prof. Tamburini, general secretary, and Prof. De Sanctis, vice-general secretary. There will be four Sections: 1. Psychology in its relation to anatomy, physiology, etc. President, Prof. Marro. 2. Introspective psychology in its relation to philosophy. President, Prof. Ardigo. 3. Pathological psychology. President, Prof. Morselli. 4. Criminal psychology and sociology. President, Prof. Lombroso. All communications should be addressed to Prof. Sante de Sanctis, Institute Fisiologico, Via Depretis, Roma.

IN MEMORY OF DR. SERAFINO BIFFI.

Dr. Serafino Biffi, who accomplished so much in the domain of psychiatry in Italy, left a lasting impression on his compatriots. A library has just been founded to commemorate his life and work.

The library is named *Biblioteca Psichiatrica Biffi* and is connected with the *Ospedale Maggiore* of Milan.

PROF. GILLES DE LA TOURETTE.

Prof. Gilles de la Tourette died after an illness of a few years' standing. Every neurologist and psychiatrist is familiar with the vast amount of work Prof. Gilles de la Tourette contributed to the study of neurology and psychiatry. His name is familiar to the profession and in medical literature, yet he was only 48 years old at the time of his death. He was an incessant worker, an excellent clinician and a brilliant writer.

DR. WEIGERT.

Dr. Weigert died August 5, 1904, at the age of 59 years. His scientific works are familiar to us all. He was an eminent pathologist and microscopist and the Weigert method in microscopy has become classic. Regardless of his great achievements in science, he was not permitted to be officially Professor of any University in Germany: he was a Jew. His laboratory in the Senckenberg Institute, however, was always crowded with zealous and admiring students from all parts of the world.

TRANSLATIONS AND ABSTRACTS OF CURRENT LITERATURE.

The Journal of Mental Science, October, 1904:

1. The Psychology of Hallucinations.—DR. W. H. B. STODDART: It is demonstrated by familiar facts that practically there is no psychological difference between perception, ideation, illusion and hallucination. While in perception and illusion there is a stimulus to the peripheral end-organ, in ideation and hallucination there is no such stimulus; in perception and illusion (in the case of visual phenomena) the stimulus to the angular gyrus arrives by way of the optic radiations, occipital lobe and occipito-angular association fibres; but in the case of ideation and hallucination the stimulus reaches it by way of other

association fibres than the occipito-angular bundle. Confirmation of this proposition is afforded by the existence of visual hallucinations in the blind, auditory hallucinations in the deaf, etc.

In the case of hallucination of vision there is a negative as well as a positive side to the process. The positive side is that the patient sees the hallucination image; the negative is that he does not see objects in the neighborhood of the image. The obvious interpretation of the negative part of the process is that the neurones that normally conduct sensations from the retina to the angular gyrus are dissociated from one another (perhaps by the retraction of the gemules); but the same effect may be produced by the patient closing his eyes. Acute maniacs frequently keep their eyes closed in order to encourage the formation of pleasant visions: in such cases the dissociation factor is presumably wanting and has to be artificially supplied by the patient. Similarly, acute maniacs frequently keep their hands over their ears in order to favor pleasant auditory hallucinations, but this is unnecessary in most cases. The author has known two patients whose auditory hallucinations were unceasingly present; he had to shout to each of them in order to make them hear his voice. Both these patients recovered and they were not deaf when the hallucinations ceased. The positive side of the hallucination process is due to stimuli reaching the centre by way of association fibres other than those by which sensations are transmitted from the peripheral sense-organs.

The hallucinated state is also favored by the absence of sensations of other modalities than that affected. The absence of other stimuli allows the affected sensory area to dominate consciousness. Hallucinations depend upon two factors, diminution of sensation and disturbance of association. Thus, the author has found that the majority of cases suffering from the hallucination known as the epigastric sensation present vast areas of analgesia. Epigastric sensation in these cases is a misnomer, as such sensations do not always occur in the epigastric region. Sometimes the uneasy sensation is referred to the umbilicus, hypogastrium, external genitalia, etc. "Globus hystericus, neurotic spine, hysterical hip, hysterical shoulder," etc., are of a similar nature. Epigastric and allied sensations are due to some affection of the cerebral cortex characterized by loss of sensation in the peripheral parts of the body. The duration of this loss may, in some cases, be limited to a short period of time. That part of the body-surface which does not suffer from loss of sensation has an abnormal amount of function thrown upon it and hallucinations may proceed from that area.

The author says that his reasoning is further illustrated by the epileptic aura in epilepsy. He says that the first symptom of an epileptic fit is loss of consciousness; in consequence of this the patient falls. It is commonly said that he loses all sensation because he is unconscious. This does not represent the true state of affairs; the truth is that the patient is unconscious because he has lost all sensation. The author says that the loss of sensation begins at the periphery (arms and legs) and allows the epigastrium to dominate consciousness; hence the aura. The last event, as the patient falls, is loss of sensation in the abdomen. This is but a special instance of the hallucinated state being favored by the absence of other sensations than those contributing to the hallucination. Therefore, the essential factor of hallucination is its negative factor. This is its essential difference from perception, ideation and illusion. There is no psychological difference between these processes.

2. Histological Studies on the Localization of Cerebral Function.—DR. ALFRED W. CAMPBELL: There is a direct correlation between physiological function and histological structure of the various territories and layers of the cerebrum. This is proven by serial studies of brains of cases of amyotrophic lateral sclerosis, of amputation of extremities and of deprivation of the special senses. In addition, a comparative examination of the cortex of two members of the anthropoid ape family and of several of the lower animals was made. The motor area is mainly characterized by giant cells of Betz, and is confined to the precentral gyrus; it does not spread to the post-central gyrus. Examination of brains of cases of amyotrophic lateral sclerosis proves this to be correct. In this disease, limited in its affection to the muscles and the motor system of the neurones, the profound changes were confined to the precentral or motor area exclusively, consisting essentially of a destruction of the cells of Betz. The author's studies show that we have certain erroneous ideas on territorial brain localization, and his histological findings seem to point to a more thorough localization. For instance, it is shown that the area for primary automatic movements is represented in what he terms the precentral area, while the centre of higher evolutionary movements is in an immediately adjoining zone that he terms intermediate precentral. Broca's centre for speech and the cheirokinesthetic centre for writing are in this zone. In the human brain the number of giant cells is far greater in the leg than in the arm area, while in quadrupeds the supply seems to be approximately equal in the respective fields. While the

skilled movements of the leg are few they are only in abeyance; they are potential and capable of development; this is demonstrated in cases of "armless wonders," whose toes and feet are educated to perform with as much skill as are our hands and fingers.

Histologically the frontal lobe is divided by the author into a frontal and prefrontal area. There is no abrupt line of demarcation between the two; but proceeding from behind forwards there occurs a step-like numerical and volumetric reduction in the cells and fibres; the reduction reaches such a marked degree in the prefrontal area that the author considers it as being extremely weak structurally—weak in cells and poor in fibres. Physiologically this area is irresponsive to electricity, and psychologically its destruction is followed by no noticeable permanent effects. He thinks, therefore, that it cannot possess great functional importance. It may be a part with a future, but at present its evolution is incomplete. It cannot be determined histologically whether the frontal lobe is performing psychic function of a higher nature; it can only be noted that the frontal lobe is more extensive in the human being than in the ape.

Some claim that the frontal lobe has an important psychic function because in cases of dementia, particularly of general paralysis, it shows atrophy—along with involvement of other centres. The author, however, explains the atrophy by the fact of the extremely weak and collapsible framework of nerve-fibres of this centre. The central gyri, the occipital and other parts do not present this naked-eye change, because they are built on a stout framework.

There is a profound structural difference between the ascending frontal and parietal lobes, and it is a mistake to suppose that the motor function is represented behind the fissure of Rolando. The post-central or ascending parietal gyrus is the main terminus for common sensory impressions. In three cases of uncomplicated tabes the author found profound changes, consisting chiefly of destruction of the large pyramidal elements limited to the ascending parietal cortex, particularly that on the Rolandic wall. Pure tabes, as is known, is an affection closely restricted to the sensory neurones. Common sensation and its simplest components are localized in the "post-central" area. Ultimately the area for common sensation will probably be divided into different centres. The parietal lobe is the seat of many common sensations. As consciousness is built on the interpretation of sensory impressions, the evolution of the parietal lobe goes hand in hand with higher development. This fact is verified on animals of

different evolutionary degrees. The temporal and frontal convolutions undergo equal expansion in the progress of phylogenic development. In three brains taken from idiots all the frontal and all the parietal convolutions are in a state of microgyria.

The Problem of Consciousness in Modern Psychology.—

PROF. SANTE DE SANCTIS: It is said that the word consciousness is given thirteen different interpretations. Yet we all know what it means to have consciousness. The difficulties are many, however, when we try to determine at what stage of development of matter consciousness made its appearance, where the seat of consciousness is in man and in animals. On the answer to these questions depends our knowledge as to whether or not there is a fetal soul, a vegetable, cellular and protoplasmic soul, a soul of crystals, of chemical affinities and a cosmic soul. Some of our predecessors claimed that none but man was endowed with consciousness, and Malebranche thought that even the higher animals were devoid of consciousness, saying of a dog that was howling with pain: "ça ne sent rien." More recent thought leads to the conclusion that consciousness is nothing but a luxury: it is intermittent in function and during its repose the psychic processes continue their function just the same. Consciousness is a subjective phenomenon and we know when we are conscious. In others, however, we recognize consciousness indirectly; one of the objective signs that enables us to do this is movement. Thus, we judge that the protozoa and metazoa have intelligence because they react to stimulation by coordinate movements with a view to conservation of the individual. Coordinate movement, however, is not necessarily a sign of intelligence or of life. While a dog, the brain of which has been removed, enacts coordinate movements, it is difficult to prove that the animal's consciousness remains intact at the same time. Yet the contrary is quite as difficult to prove. Another objective sign of consciousness designated by some may be called morphological: consciousness, according to some, is intimately related to the nervous system; wherever there is a nervous system, they claim, there is consciousness and *vice versa*. The author does not agree with this reasoning: under these conditions consciousness seems to be synonymous with complexity or coordination of motor functions. Others claim that a criterion of consciousness is memory: those who have no memory have no consciousness.

The author thinks that probably there are minima and maxima of consciousness, being highest in man and almost indistinct in the being at the lowest step of the zoological scale. For this reason psychology should not confine itself to the study of complete and perfect consciousness exclusively, but should also delve into the study of partial, dim as well as sub-consciousness. There is nothing positive to warrant our saying that there is no cellular or even mineral consciousness. The latter may be qualitatively different from ours, and it is allowable to think that all the lower consciousnesses are united and represented in man,—although it is difficult to imagine that our consciousness should represent the resultant of different consciousnesses. While it is of no practical value to search for the dimmest consciousness found in animals below the primates, it is not justifiable to reject as impossible the psychology of animals.

Consciousness cannot be localized in any special part of the human body. It is intimately connected with the nervous system and, to a certain measure, to the entire organism. Consciousness is principally a function of connection. There is nothing to warrant the conclusion that the cerebral cortex is the exclusive seat of consciousness. If the medulla and spinal cord do not manifest conscious acts in man, it is because the cerebral cortex presides over the function of the other nervous organs. In some animals, however, we find that the ganglia, the peripheral cells and perhaps even the epithelial cells may represent the source and seat of consciousness. All that can be said of the seat of consciousness in man is that the cerebral cortex constitutes principally the somatic parallel of the phenomenon of clear consciousness (*Archives de Psychologie*, tome III, No. 12, 1904).

Researches in the Chemical Changes in Dementia Precox. —DRS. ANTONIO D'ORMEA and FERDINANDO MAGGIOTTO: These researches are urological exclusively, dealing with the phosphates, sulphates, chlorides, urea and uric acid. There is a marked difference between the amount of these ingredients eliminated in the normal subject and in those suffering from dementia precox. The total amount of urine excreted during the twenty-four hours is one-fifth less than normal and the specific gravity is decreased, showing a decreased amount of the solids. The amount of nitrogenous compounds is greatly diminished. The amount of urea eliminated is about one-half as compared with the normal and the amount of uric acid eliminated is one-third of the normal. The

inorganic elements are also decreased to more than one-half the normal; this is especially noted in the amount of phosphoric acid. The decreased amount of sulphuric acid is slightly less, but quite marked. The amount of chlorides, on the contrary, is increased. While it is difficult to say of what importance these showings are in connection with the pathogenesis of dementia precox, the constance of occurrence of these facts are worthy of note. These facts are so much the more interesting because the peculiar slowness of renal elimination in dementia precox has been demonstrated by the authors in a previous paper: there was markedly retarded renal elimination of methylene blue and of iodide of potash as compared with the normal elimination; the total amount of these substances was eliminated after a much longer period than usual. The organic catabolism in dementia precox is considered, therefore, to be markedly decreased. This result has been constantly obtained in dementia precox in all its stages,—during the periods of mental torpor, passive catatonia or excitation. The constancy with which these findings are obtained in dementia precox indicates that the facts mentioned above are proper to dementia precox (*Manicomio provinciale di Ferrara*, 1904).

The Reflex Action, by Way of the Dura Mater, on the Pneumogastric Nerve as One of the Regulators of the Cerebral Circulation.—DR. IOGANSO: It is well known that an increased blood afflux to the brain increases the intracranial pressure and that this is immediately followed by slowing of the cardiac beats. Temporary weak cerebral circulation, on the contrary, is followed by decreased intracranial pressure and more frequent cardiac beats. There is no satisfactory explanation of these facts. According to Franck, the increased cranial pressure itself acts as an irritant on the pneumogastric nerves and inhibitory phenomena are results thereof. The author does not accept this explanation because it cannot reasonably be admitted that the nucleus of the pneumogastric nerve is, under the conditions considered, compressed to the exclusion of all other nuclei similarly situated. His own explanation is that the regulation of the cerebral circulation depends on a special reflex enacted, by way of the cerebral dura mater, on the vagus nerves. The dura mater is supplied by filaments from the *rami recurrentes Arnoldi*, the 1st, 2d and 3d branches of the trigeminus and *meningeus vagi*. The inner surface of the dura mater is in close contact with the brain and irritation quickly causes reaction by pain, etc. A temporary increase in size of the

brain presses the dura mater against the inner surface of the skull and on the nerve endings of the dura mater. This irritation brings about the reflex action, through the *rami recurrentes trigemini* and the *meningei vagi*, on the vagus nerves. All the ramifications of the trigeminus are intimately connected with the inhibitory tracts of the vagus and a reflex act of the vagus can easily be provoked through the trigeminus. The author then concludes that his hypothesis of the reflex here considered is highly justifiable. He says that his hypothesis is supported by the conditions of the pulse in meningitis, the numerous cerebral apoplexies during the course of general paralysis—when the dura mater reflex is considerably impaired by pathological conditions and that many other facts support his view (*Vestnik Dushvernikh Boleznei*, No. 6, 1904).

Comparative Psychiatry.—KRAEPELIN: The study was made in Java. The tropical climate has no especial effect on the European. The effect of alcohol on the latter presents nothing special. The natives, who are Mussulmans, do not drink alcoholic beverages, but make up for this by intoxicating themselves with opium. They do not present any psychoses due to opium. The manifestations due to excessive use of opium are less severe than those due to morphinism. Suspension of the use of opium is not characterized by any special symptoms. The use of the root of betel has no special action either on the body or on the brain. Among 370 native insane the author found neither general paralysis nor cases of cerebral syphilis. Among 50 European insane, on the contrary, there were 8 of these cases. The demential processes manifest themselves as they do in Europe. Mani-depressive insanity is more rare than in Europe, while epilepsy is of more frequent occurrence. In mani-depressive spells the depressive phase is less frequent of occurrence and ideas of culpability are not in evidence. The spells of excitation are more violent. In dementia precox the phase of initial depression is wanting, the delusions are rare, the catatonic symptoms are not marked, profound stupor and complete dementia are rare of occurrence. A special affection termed *Laffa* is characterized by movements resembling automatic movements and by coprolalia. *Amok* is not a clinical variety; it is characterized by various excitations with a tendency to commit acts of violence. It may take place in catatonia, epilepsy and other affections (*Archives de neurologie*, November, 1904).

To Think and to Know One's Self.—PROF. SANTE DE SANCTIS deplors the facts that education of to-day is directed exclusively almost towards the training of our sensory sphere while the intellectual sphere properly speaking is left to take care of itself the best way it can. While he approves of the training of the young in the matter of understanding their respective outward worlds, he thinks that after childhood and adolescence it should be our particular task to train our minds for introspection that alone enables one to study and understand one's self. Mental gymnastics are not only wholesome and useful to all individuals, but also yield excellent moral riches that become our main pillars of support in moments of moral crises,—in those moments where no kind of sport can play the rôle of a sedative to our shattered sensibilities. Men and women of to-day are more than ever in need of mental training that teaches the proper method of introspection. A few instances are cited of the beneficial effects of introspection exercised by suggestion among the neuro- and psychopaths. If good results can be obtained from proper introspection among the sick, the author says, how much better results could we not obtain among the well—if they were taught in proper time to practice wholesome introspection? We energetically teach the young to know the external world, and it is proper to develop their sensorial spheres. It is most urgent, however, not to stop there and to teach persons of all ages to also know their inner worlds (*Rivista d'Italia*, August, 1904).

An Unusual Case of "Idiocy."—PROF. KOVALEVSKI reports a case of so-called idiocy that was due to peculiar circumstances: the subject, a boy, 14 years of age, was brought to the Kharkov Hospital for the Insane with claim for admission there because he was an idiot. The boy answered the description of an imbecile at all events: he did not know the names of common things about him and his vocabulary was quite limited; he could not read, write or count and did not know coins of money or the use thereof. He remained silent, alone in some corner, was filthy and evacuated his bowels and bladder in the presence of those about him in the room. The history of the case revealed the fact that the boy had never come in contact with human beings except with his father. The latter was a shepherd and had the boy with him for a short while. He seldom spoke to the boy and allowed him to lead a dumb existence in a place where no human beings ever disturbed their monotony. In the hospital the boy was given a course of instruction. During one year he learned as much as children of his age and social station generally know, and after the second year

he could read and write and left the hospital to assume the duties of a paid employé. This kind of "idiocy" is considered by the author as a disease of society (*Vestnik Dushevnikh Boleznei*, No. 6, 1904).

For the Backward and Abnormal Children.—According to the French law of March 28, 1882, primary instruction is obligatory for all children between the ages of six and thirteen inclusively. This law has not been carried out in the case of the defective children, especially among the blind and the deaf and dumb, a large number of whom are handled by the Jesuits. The approximate number of the deaf and dumb of the ages mentioned is seven thousand and that of the blind is between four and five thousand. Popular sentiment and reasoning has thus far succeeded in relegating one-third of the deaf and dumb and four-fifths of the blind of school age to the hands of charity and has thus deprived them of their right to obtain proper instruction. Economically this is extravagance and morally the law has been violated. The Minister of Public Instruction, M. Chaumié, has appointed a large committee of distinguished men to report to him on this subject. Dr. Bourneville, the distinguished psychiatrist and friend of the backward children, is one of the members of this committee (*Archives de neurologie*, November, 1904).

On the Question of Cerebral Overwork in Schools.—DR. KLEVZOV: Many cases of neuroses and psychoses are due to excessive mental strain of school children. This is especially the case among girls whose over-zealous teachers are generally women. An example of a part of a day's lesson for girls between 9 and 10 years of age, in the *Moscow Gymnasia*, is as follows: for the lowest grade, reviewing 25 pages of geography, memorizing ten lines of French; for the grade above, fifteen histories from the Old or New Testament, including numerous biblical proper names, fifty lines of difficult poetry to be learned by heart, etc. Some of the women teachers are said to be pedantic in their requirements and give low marks to their unfortunate victims if the lesson is learned imperfectly. Some of the little girls thus tutored have been treated by the author for hysteria, chorea and other disturbances due to excessive mental overwork (*Vestnik Dushevnikh Boleznei*, No. 6, 1904).

Contribution to the Study of the Functions of the Optic Thalamus.—DR. BENAKY: Recent investigations have demonstrated that the mimic movements of the face

are controlled by the optic thalamus. The author reports a case in support of this view and the autopsy of the case revealed an extensive tumor that involved, besides other portions of the brain, the optic thalamus. Among numerous symptoms that corresponded to the cerebral lesions the author lays stress on the particular menacing attitude of the patient during life: no matter what was said to her, she replied "I shall strike you." This menace was accompanied by an appropriate facial expression and she attempted every time to carry her threat into execution. The extensive lesion of the optic thalamus found at the autopsy is considered of significance in connection with the special symptom mentioned. Some cases are cited in which a similar lesion corresponded with spells of irresistible laughter (*Archives de neurologie*, November, 1904).

The Mode of Action of Abdominal Massage on Arterial Hypertension.—DR. F. CAUTRU: The author demonstrates by graphic illustrations the following conclusions:

Abdominal massage is curative in transitory hypertension (premenstrual migraine, pseudo-angina pectoris, toxialimentary dyspnea, chlorosis, etc.). Massage is also a preventive measure in permanent hypertension (angina pectoris, etc.) and in confirmed arterio-sclerosis. Diuresis is augmented (through action on the splanchnic nerve), renal elimination of poisonous elements is facilitated and osmosis is augmented. This favorable effect is produced through reflex action (*Archives de Médecine*, May 17, 1904).

Cases of Cerebral Pseudo-Tumors with Recovery.—DR. NONNE: Twelve cases are reported. They presented the symptomatology of cerebral tumors, of sub-acute or chronic nature with papillary stasis. Eight cases recovered (after 2½ and 3½ years) and four ended in death. None of them suffered from hydrocephalus or tuberculosis. One of the cases died suddenly after he had been considered as cured during a period of two years. In the other three cases the autopsy was negative. There is an analogy between these cases and those of Jacobson of hemiplegia without anatomical lesion (*Archives de neurologie*, November, 1904).

The Insane at Large: In a fit of delirium, a certain E. Z. overpowered all the members of his family and killed his

sister, father and mother. His brother-in-law, who succeeded in making his escape, soon returned with police officers, and the patient was found fast asleep among the dead in a pool of blood that came from their wounds (*Archives de neurologie*; November, 1904).

Leonardo Da Vinci and Cerebral Localization.—

PROFESSOR SANTE DE SANCTIS, in *Archives de Psychologie*, tome III, No. 12, 1904, makes mention of Leonardo da Vinci's opinions on the localization of the mental faculties in man. Leonardo da Vinci thought that the mental faculties were located in the cerebral ventricles. Impressionability, that corresponds to the perceptive faculty, he located in the lateral ventricles; in the middle ventricle he located the centre of general sensibility and the centre of memory he located in the fourth ventricle. Leonardo da Vinci's manuscript was never published. Prof. De Sanctis saw a photograph of it in the Royal Library, at Windsor.

Two Cases of Merycism.—DRS. RAVIART and CAUDRON: One of the cases was a melancholiac with hallucinations of hearing and the other an idiot. Both had bad teeth. They experienced great pleasure in eating gluttonously, in regurgitating the food they had swallowed and chewing it over and over again. Interesting remarks are made about the appearance of the various foods after regurgitation (*Archives de neurologie*, November, 1904).

BOOK REVIEWS.

Acute Paranoia (Paranoia Acuta). The Clinical Side of the Question.—DR. P. B. GANNOUTCHKINE: Lissner and Gheshel, Moscow, 1904. As is indicated in the title of this work, the clinical side of the question only is considered here. In the introduction, the author makes a review of the opinions of various authors regarding the question of paranoia. Some authors claim that acute paranoia may be excluded from the classification of mental diseases, some disclaiming the existence of acute or periodic paranoia, while others give detailed descriptions of acute paranoia as a distinct and individual psychosis. Paranoia, as defined by some, belongs to the class of psychoses in which the intellectual sphere is involved, the psychoses in which the emotional sphere is

involved constituting a different class (mania, melancholia). In the class of psychoses with involvement of the intellectual sphere may be grouped: ania acuta, amentia of Meynert or dysnoia acuta, and paranoia acuta. Chronic paranoia belongs to the same class and is distinguished by its chronicity. The author considers acute paranoia as a distinct psychosis. The main bulk of the work is devoted to the consideration of acute paranoia as a clinical entity. Various chapters are then devoted to the consideration of symptomatic acute paranoia, idiopathic acute paranoia, periodic paranoia, cases of acute paranoia published as such, cases of acute paranoia collected by the author, and clinical data on acute paranoia. Chronic and intermittent paranoia are also considered in connection with the main object. Recovery from chronic and secondary paranoia is also analyzed. The author feels satisfied that paranoia may be considered as a special clinical entity that may occur either by itself or in conjunction with some other psychoses. It has been demonstrated similarly, he says, that Korsakoff's disease may manifest itself not only during the course of polyneuritis but also in conjunction with senile dementia, cerebral syphilis or edema and even during the course of progressive general paralysis. The same is true of acute delirium: it may occur during the course of various diseases. Catatonia is also a clinical entity that may be found in conjunction with different psychoses. This manner of viewing psychiatric facts has been fruitful and has led to a better interpretation of established clinical forms of diseases. The latest effort in this direction of determining psychiatric entities is seen in the grouping of paranoia as a clinical entity.

Lesions du Cerevelet Chez les Paralytiques Généraux et les Déments.—DR. PAUL JEANTY, Thèse, 1903.—The cerebellum is always involved in general paralysis to a greater or lesser extent. The lesions may be infectious, parenchymatous, interstitial or vascular. The parenchymatous lesions involve all the elements of the cerebellar cortex. The Purkinje cells are most frequently involved, because of their functional importance. The Purkinje cells may be found in various conditions, beginning with the normal state and ending with complete disintegration. The lesions of these cells are far more marked in general paralysis than they are in dementia during the course of psychoses in general. In general paralysis the cellular disintegration is complete, rapid and diffuse. The lesions reach a

minimum degree in sensory ataxia. In the usual dementias the disintegration is slow of development, the cells appearing rather as atrophied and vitally reduced than disintegrated elements.

The amount of the disintegrated cells is marked, so that the number of normal cells is only about one-tenth or one-twelfth of the normal. The cellular layer is wasted and its elements are rare. The central ganglia are less affected than is the cortical substance. The fibres are often degenerated and sparse. Neuroglia proliferation is found in the interstitial tissue. In some cases may be found independent vascular alterations that are probably due to superadded infections.

In acute encephalitis the cerebellar cortex is almost normal. The number of disintegrated cells is almost nil and the appearance of the altered cells is quite different from that found in the dementias. In acute encephalitis the most noticeable thing is the congestion and marked diapedesis accompanied by meningeal infiltration.

The lesions mentioned have nothing pathognomonic in connection with the diseases enumerated above. The aspect of the lesions simply enables us to differentiate between acute, subacute and chronic encephalitis from that found in general paralytics and in dementes.

It is as yet difficult to draw a parallel between given cerebral lesions and their corresponding symptomatology. Nevertheless, it is of interest to note that the cerebellum is markedly affected in general paralytics presenting sthenic and static disturbances of co-ordination.

Rapports Etiologiques de la Syphilis Héritaire avec les Encephalopathies Chroniques de l'Enfance.—JEAN

JACQUES MEYER, Thèse, Paris, 1904.—The author has made a thorough study of the subject of hereditary syphilis in its relation to chronic encephalic diseases of infancy. He arrived at the following conclusions:

The various forms of chronic encephalitis of infancy are seldom due to hereditary syphilis. This condition is due to the fact that the syphilitic virus kills the fetus in utero, or, if the child is born, it succumbs a few days after birth. A child born with hereditary syphilis, therefore, has no time to manifest any cerebral lesions. Consequently, hereditary syphilis figures at the end of the list of the various hereditary intoxications that are causes of chronic encephalitis of infancy.

BOOKS AND PAMPHLETS RECEIVED.

Dr. S. Weir Mitchell.—The Youth of Washington Told in the Form of an Autobiography. The Century Co., New York, 1904.

Dr. Sergio Sergi.—Le variazioni dei sulchi cerebrali e la loro origine segmentale nell' *Hylobates*.

Dr. Donald M. Barstow.—A Case of Supposed primary Tuberculosis of the Pharyngeal Tonsil.

Dr. S. Carpenter.—Treatment of Anemic Conditions in Children with Iron.

Dr. Eugenio Medea.—La Patogenesi della Malattia di Stokes-Adams. Osservazioni Cliniche ed Anatomico-Patologiche.

Dr. Eugenio Medea.—Il Fanciullo Defficiente.

Dr. Eugenio Medea.—L'Idroterapia e le Scuole.

Dr. J. Guitman.—A Case of Epidural Abscess of Otitic Origin. Operation. Recovery.

Drs. Marie and Viollet.—Spiritisme et Folie.

Dr. Sinclair Tousey.—The Treatment of Tuberculosis of the Larynx and of the Prostate Gland by the X-Ray, High Frequency Currents, and the Cooper Hewitt Light. Demonstration of Special Apparatus and of a Rapid Method of Skiagraphy.

Dr. William P. Small.—Preliminary Treatment in Minor Gynecological Surgery.

V. K. Froula.—Report of Proceedings of the National Conference on Secondary Education and Its Problems. Held at Northwestern University, October 30 and 31, 1903. Published by the University, Evanston, 1904.

Annual Announcement, the Vermont Bulletin, University of Vermont, Medical Department, Burlington, Vermont. 1904-1905.

Dr. Biate.—Des maladies du sommeil et des crimes commis dans le somnambulisme.





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